THE

Volume Fifty-Four

Journal

OF THE

AMERICAN OSTEOPATHIC ASSOCIATION

Number 10 | 212 East Ohio St., Chicago 11, Ill. | June, 1955

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Published monthly by the American Osteopathic Association. Printed by Pioneer Publishing Company, Publication Office, 100 S. Kenilworth Ave., Oak Park, Ill. **EDITORIAL AND EXECUTIVE OFFICES,** 212 E. Ohio St., Chicago 11, Ill. Subscription $10 a year; single copies, $1.00. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized August 31, 1922. Entered at Oak Park, Ill., Post Office as second class matter, April 1, 1926, under Act of March 3, 1879.

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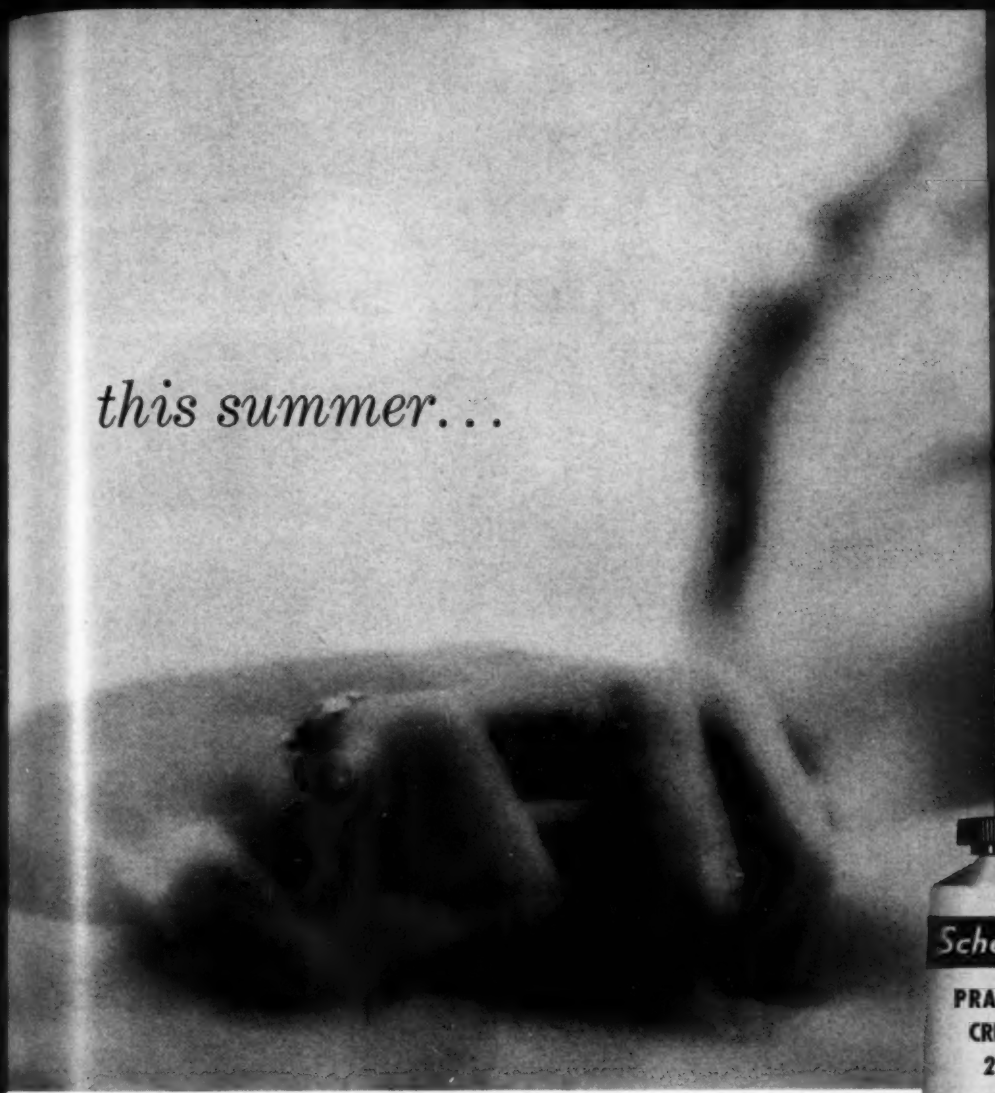

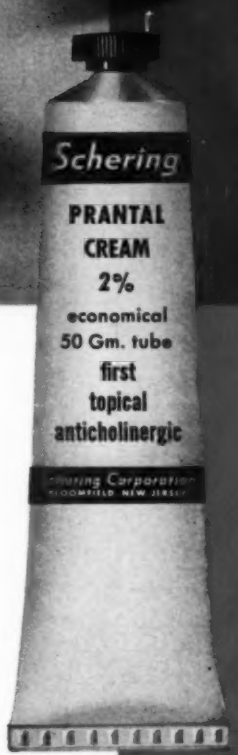

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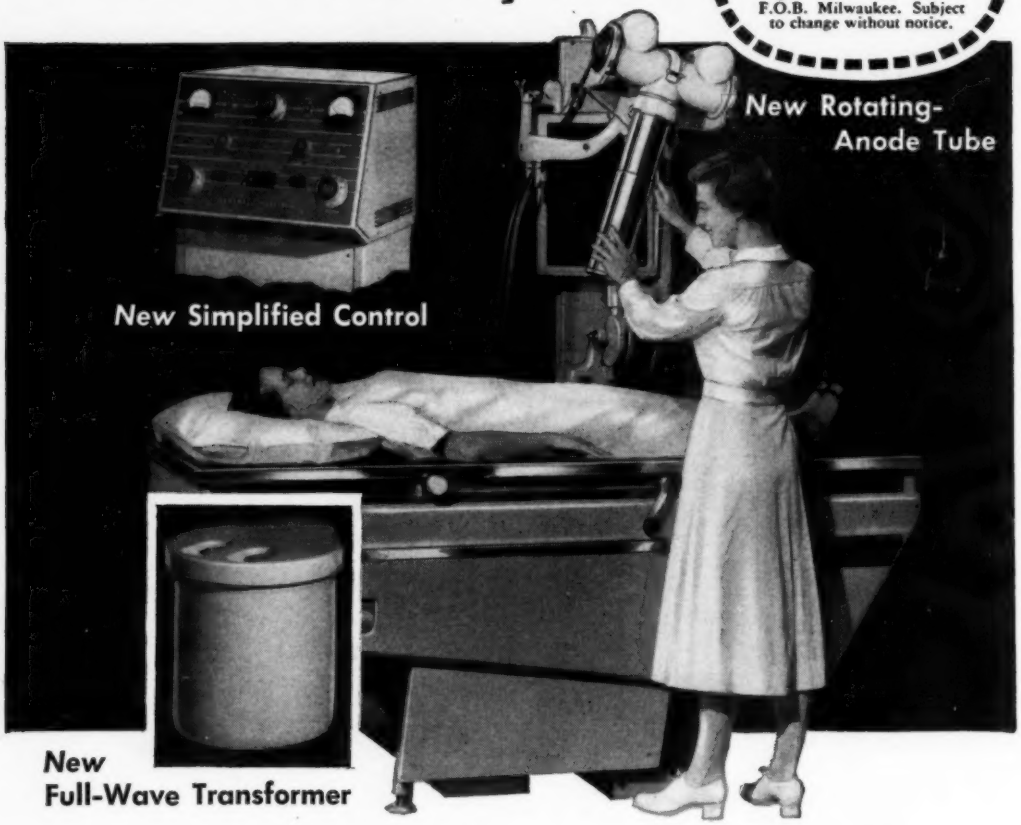

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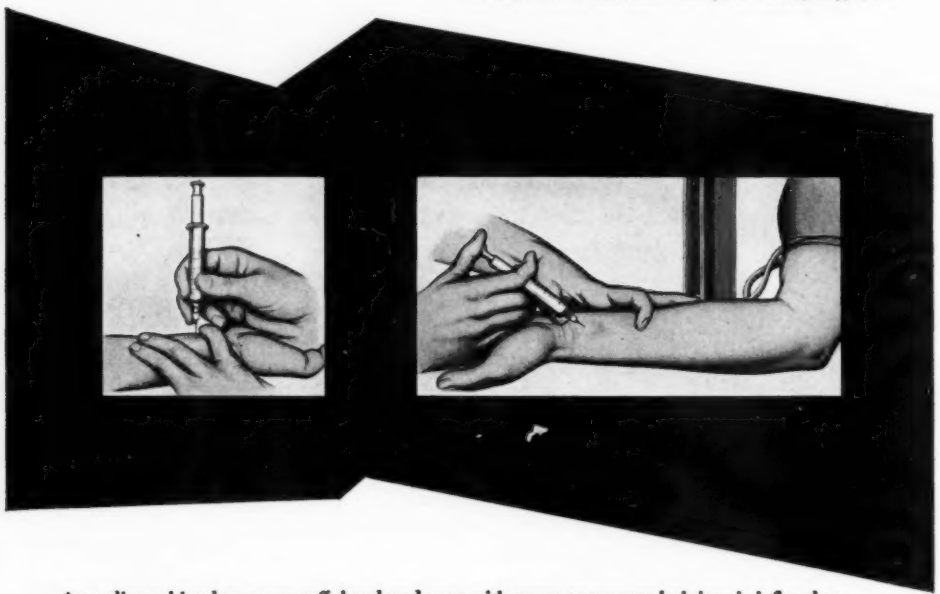

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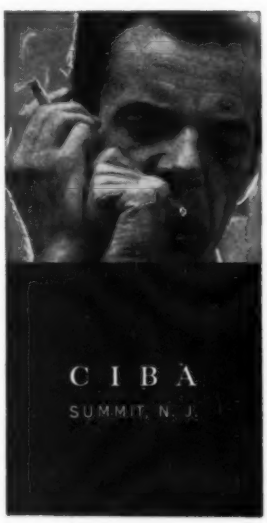

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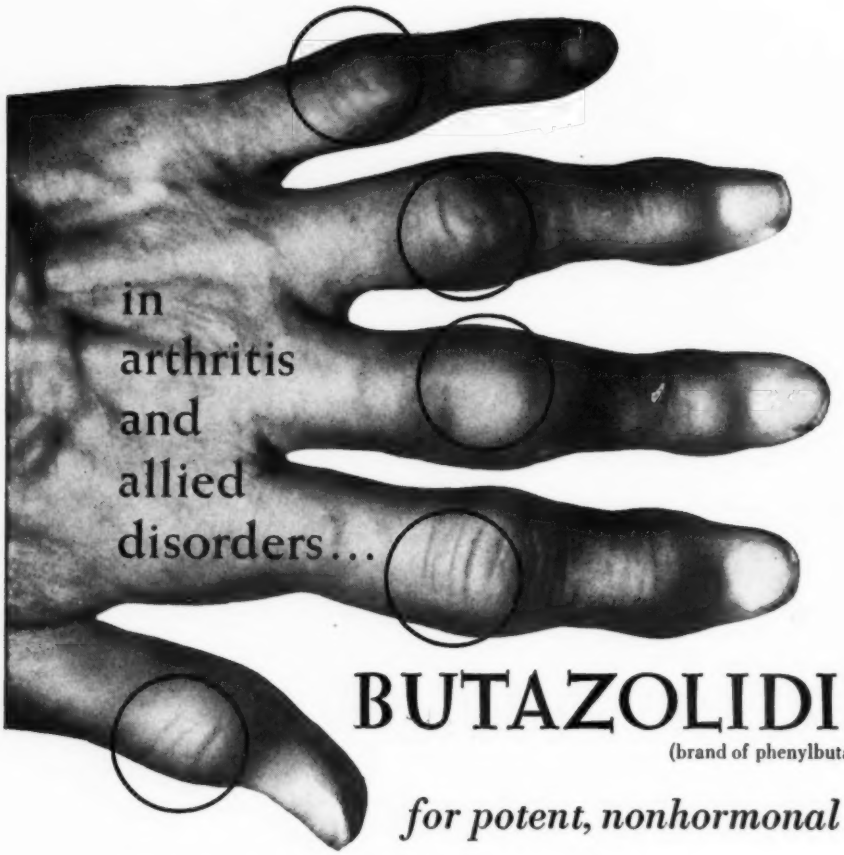

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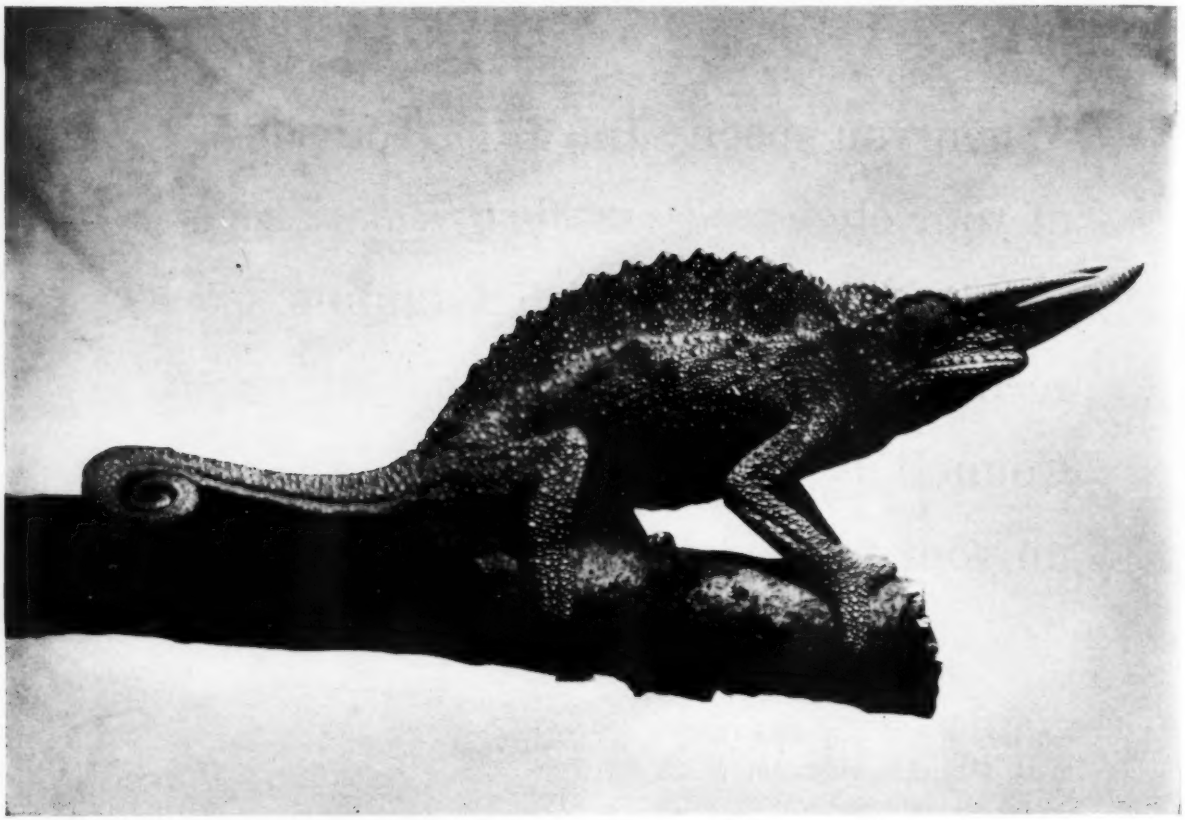

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1. MERLIS, S.: Diamox: A Carbonic Anhydrase Inhibitor—Its Use in Epilepsy. Neurology. 4:11, 863-866 November 1954.

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LEDERLE LABORATORIES DIVISION *AMERICAN Cyanamid COMPANY*
PEARL RIVER, NEW YORK

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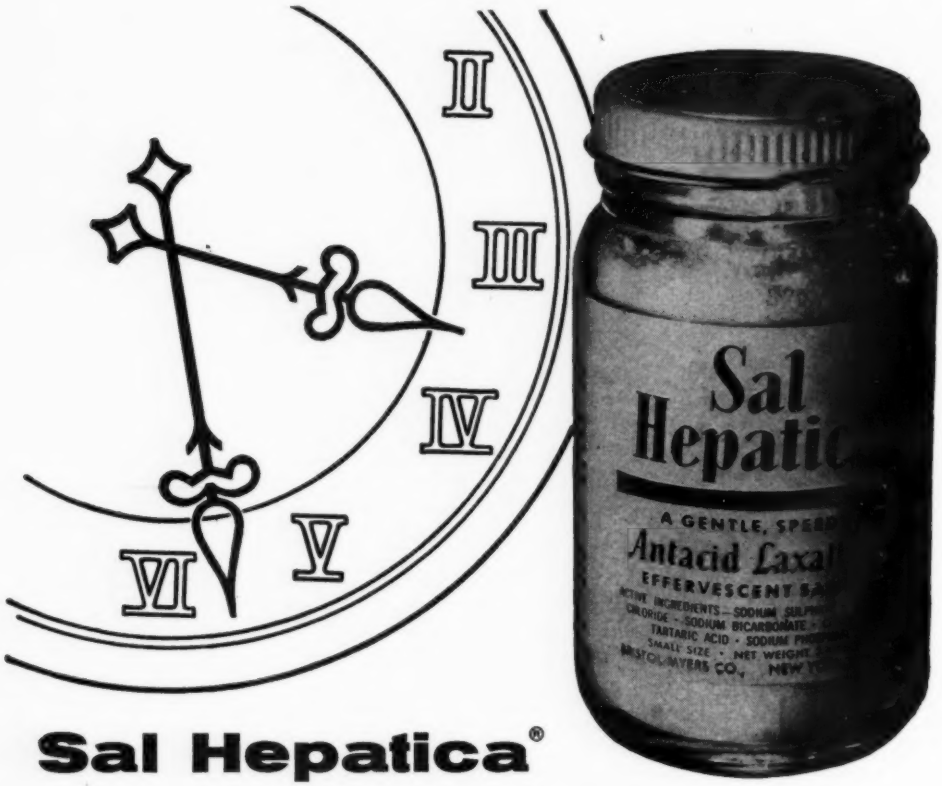

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SAL HEPATICA'S Action Has a Sound Pharmacologic Basis

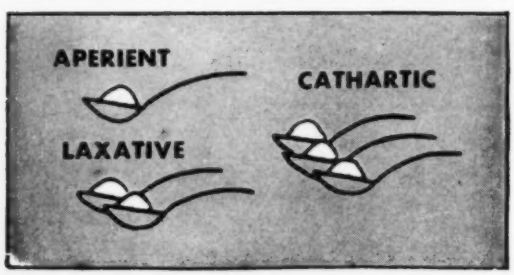
1. It is antacid and effervescent. Reduction of gastric acidity decreases emptying time of the stomach.[1]

Effervescent mixtures also shorten the emptying time.[2]

Thus SAL HEPATICA quickly leaves the stomach to enter the intestine where its laxative action takes place.

2. It stimulates intestinal peristalsis by its osmotic action. The fluid drawn into the intestine is a mechanical stimulus to evacuation, which usually follows promptly.

Prompt, gentle laxation without griping follows the use of pleasant-tasting SAL HEPATICA. The gastric hyperacidity so frequently accompanying constipation is relieved, too, because SAL HEPATICA is antacid.



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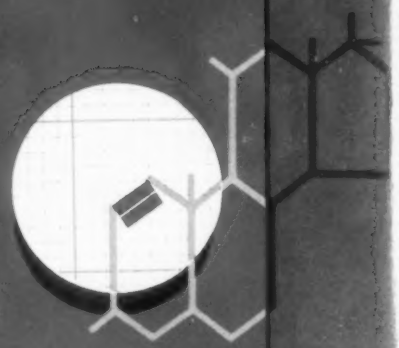

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2. Waine, H.: Bull. Rheumat. Dis. *5*:81 (Jan.) 1955.
3. Herzog, H. L., and others: Science *121*:176 (Feb. 4) 1955.

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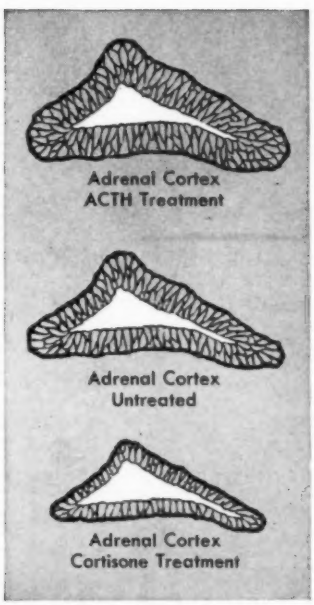
A *Yes*—it should be prevented! Atrophy of the adrenal cortex with loss of secretion of adrenal cortical hormones deprives the patient under cortisone therapy of a defense mechanism which can assume vital importance when exposed to unexpected stress.



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**Chick, H.: Nutrition 7:59, 1953; Cotereau, H. et al.: Nature 161:557, 1948. Jolliffe, N. et al.: Clinical Nutrition; Hoeber, New York, 1950; pp. 586-601.*

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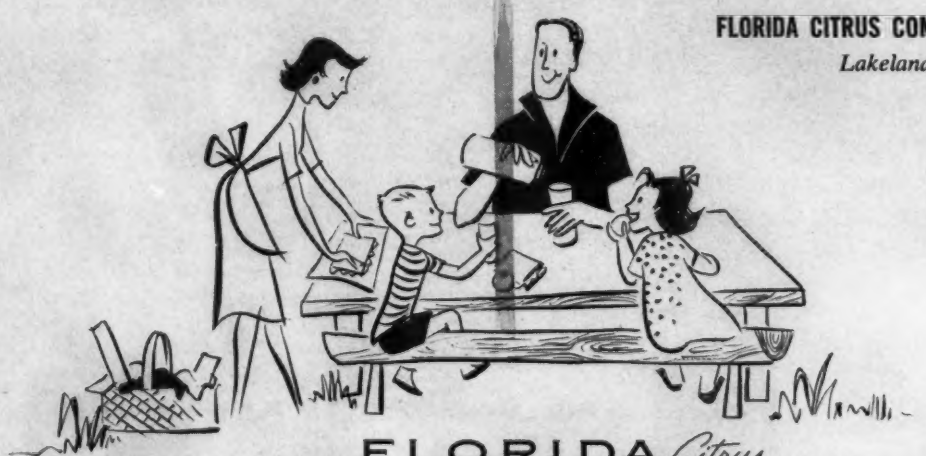

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1. Finnerty, F. A., Jr.: The Value of Rauwolfia Serpentina in the Hypertensive Patient, Am. J. Med. *17*:629 (Nov.) 1954.
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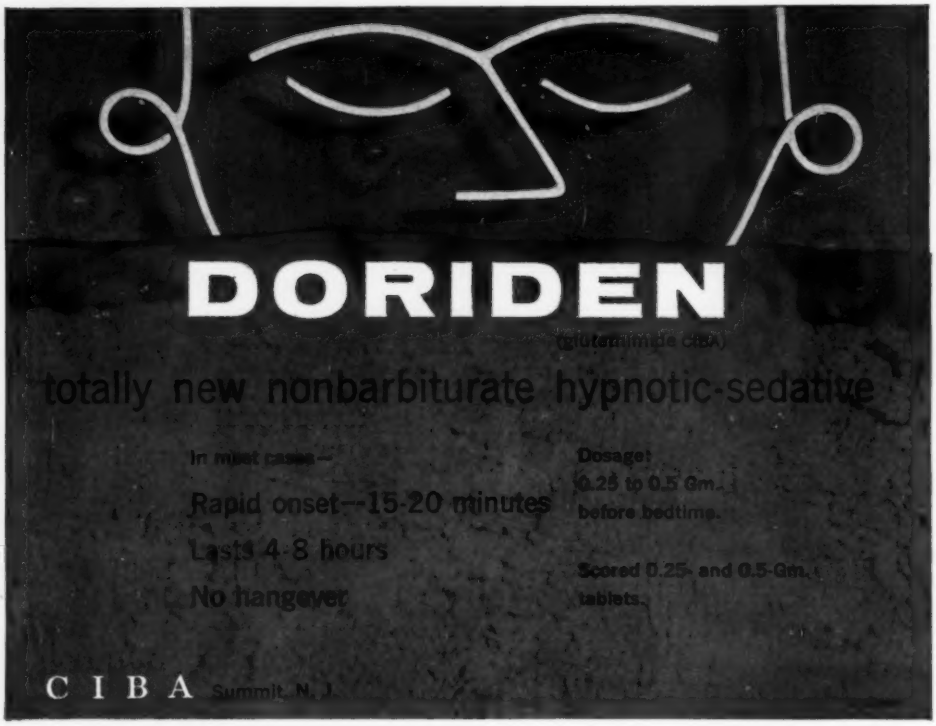

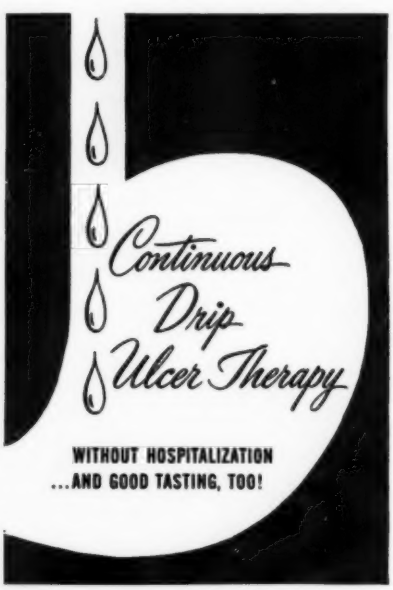

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*Steigmann, F., and Goldberg, E.: Ambulatory Continuous Drip Method in the Treatment of Peptic Ulcer, Am. J. Digest. Dis. 22:67 (Mar.) 1955.

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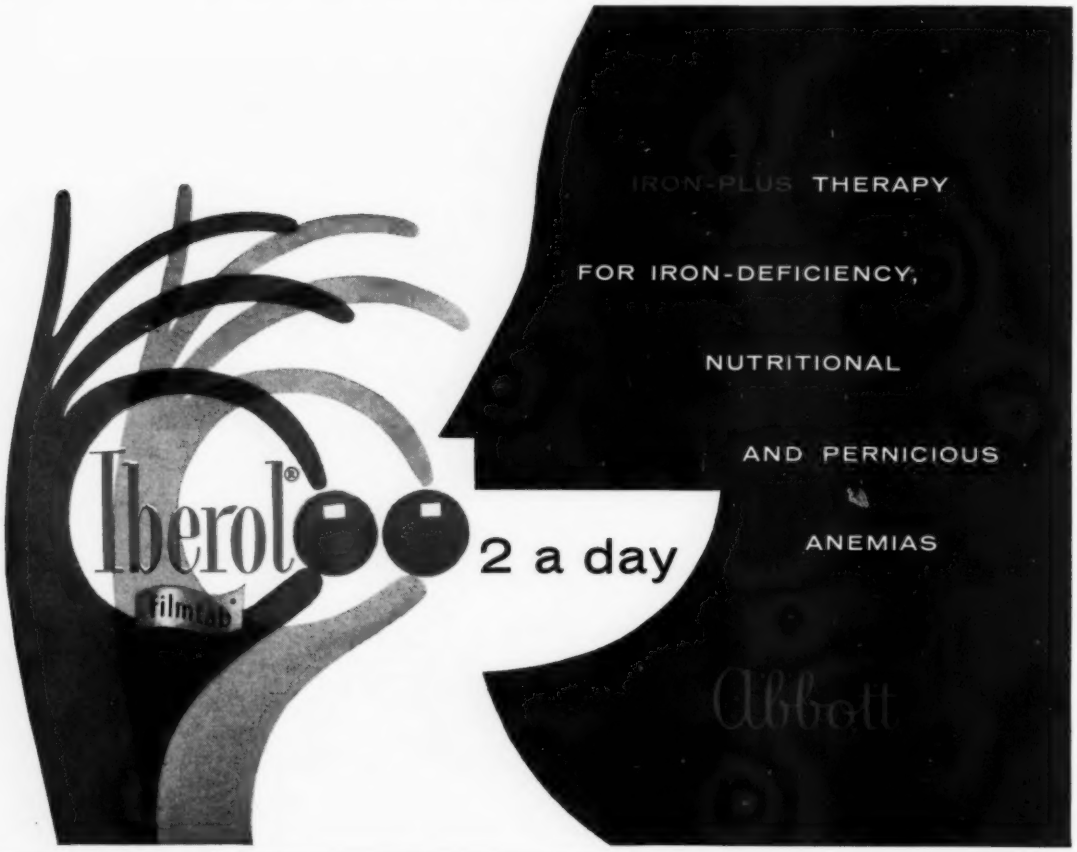

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Supplied: Tablets, 25 mg. (scored), vials of 24 and bottles of 100; also, 12.5 mg. (scored), bottles of 100. Elixir, 25 mg. per 5-cc. teaspoonful, bottles of 1 pint.

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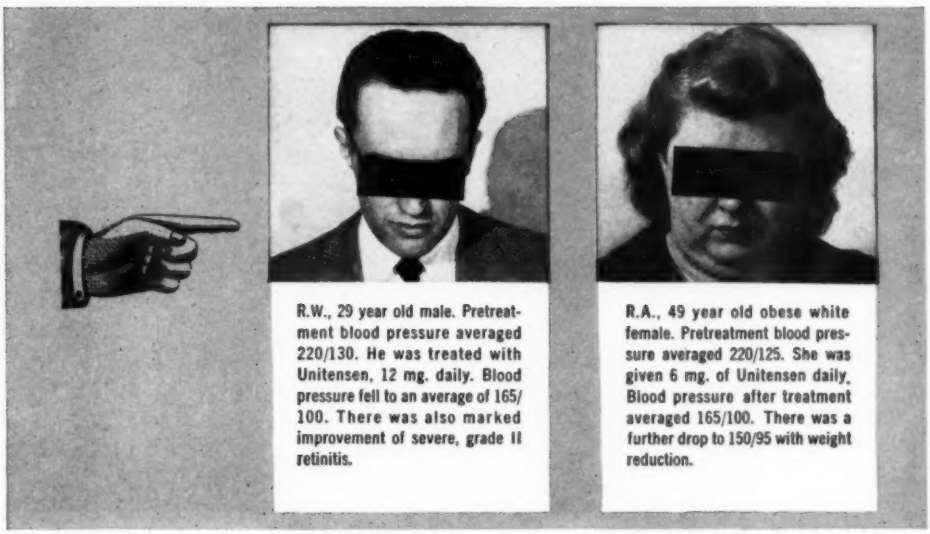

R.W., 29 year old male. Pretreatment blood pressure averaged 220/130. He was treated with Unitensen, 12 mg. daily. Blood pressure fell to an average of 165/100. There was also marked improvement of severe, grade II retinitis.

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exerts maximum antiallergic action during the period of allergic stress...

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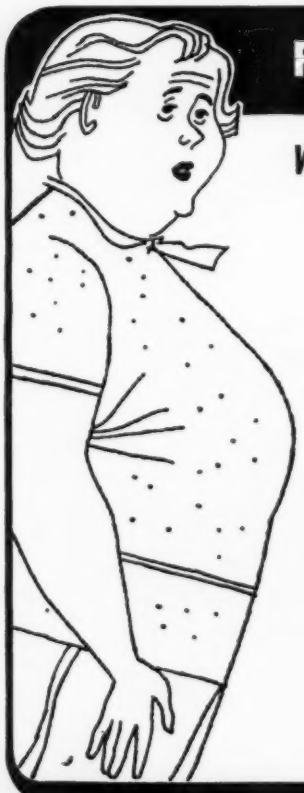

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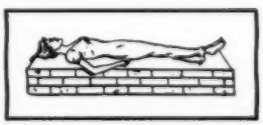

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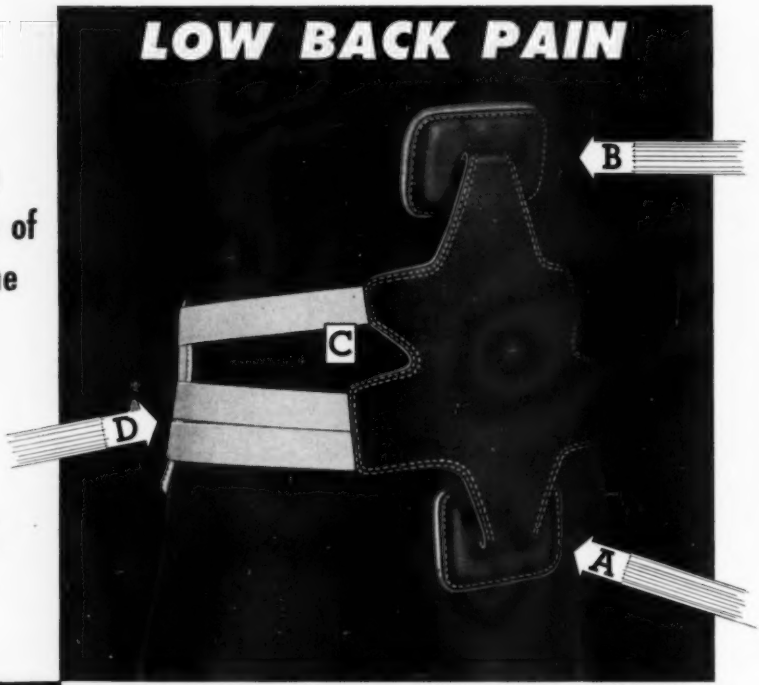

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Descriptive literature on request

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IN CONCEPTION CONTROL

prescription to fit the patient

No one method of conception control is applicable to all patients. For some, the "diaphragm-and-jelly" technic serves best, for others "jelly-alone" is adequate. Responsibility for selecting the more desirable of the two methods often rests solely with the physician. Not infrequently, if the patient is permitted to share in the decision, a higher degree of successful contraception will be attained.

From the physician's point of view: When the life and health of the patient depend upon prevention of pregnancy, the diaphragm-jelly method becomes first choice. If certain anatomic difficulties exist such as relaxed pelvic floor,[1,2] extensive cystocele,[1-3] extensive rectocele,[1-3] intact hymen,[3] short anterior vaginal wall,[2] third degree retroversion of the uterus,[2] acute anteflexion of the uterus[2] or complete prolapse,[3] proper placement of the diaphragm usually will not be feasible. Safer than jelly applied to an improperly fitting diaphragm is jelly-alone inserted into the vagina. Inability to learn the diaphragm technic also necessitates jelly-alone.

From the patient's point of view: The highly fertile multiparous patient who considers her family complete will seek the extra protection of a diaphragm. Women motivated by a morbid fear of pregnancy will prefer to reduce the risk of conception by using both a mechanical device and a spermaticide. Conversely, if there is no urgent need to avoid conception,[4,5] or if there is an unwillingness to use the diaphragm, jelly-alone will be the patient's choice because of its simplicity. Practical considerations, such as crowded living conditions or inaccessible toilet facilities may necessitate jelly-alone.

Dependability of each technic: Diaphragm-and-jelly offers the most dependable conception control,[1,5,6-8] with reliability of 95% to 98%.[6,7] Jelly-alone will provide a high degree of protection in nonparous women and in women of low parity.[4] Among 325 women who used jelly-alone (RAMSES Vaginal Jelly*) for 3 months to 3 years, the overall unplanned pregnancy rate was 16.7 per 100 patient-years of exposure. Over 35% of the women who become pregnant admitted negligence or failure to use the method properly. Thus the actual pregnancy rate was 10.82 per 100 patient-years of exposure.[4]

After the decision has been made: When the choice favors diaphragm-and-jelly, the RAMSES® "TUK-A-WAY"® kit is recommended. The RAMSES diaphragm is flexible and cushioned. It provides an optimum mechanical barrier with utmost comfort. In combination with RAMSES Jelly,* it offers an unsurpassed contraception technic. Where anatomical, psychological or economic factors indicate the use of jelly-alone, RAMSES Vaginal Jelly can be confidently prescribed. *Both products are accepted by the appropriate Councils of the American Medical Association.*

Bibliography: 1. Reich, W. J., and Nechtow, M. J.: Practical Gynecology, Philadelphia, J. B. Lippincott Co., 1950. 2. Tietze, C.; Lehfeldt, N., and Liebmann, H. G.: Am. J. Obst. & Gynec. 66:904, (Oct.) 1953. 3. Greenhill, J. P.: Office Gynecology, ed. 5, Chicago, The Year Book Publishers, Inc., 1948. 4. Finkelstein, R.; Guttmacher, A., and Goldberg, R.: Am. J. Obst. & Gynec. 63:664 (March) 1952. 5. Barnes, J.: Lancet 2:401 (Aug. 22) 1953. 6. Gamble, C. J.: Ann. New York Acad. Sc. 54:840, May 2, 1952. 7. Novak, E.: Textbook of Gynecology, ed. 3, Baltimore, The Williams and Wilkins Co., 1948. 8. Council on Pharmacy and Chemistry of the A.M.A.: New and Non-official Remedies for 1954, Philadelphia, J. B. Lippincott Co., 1954.

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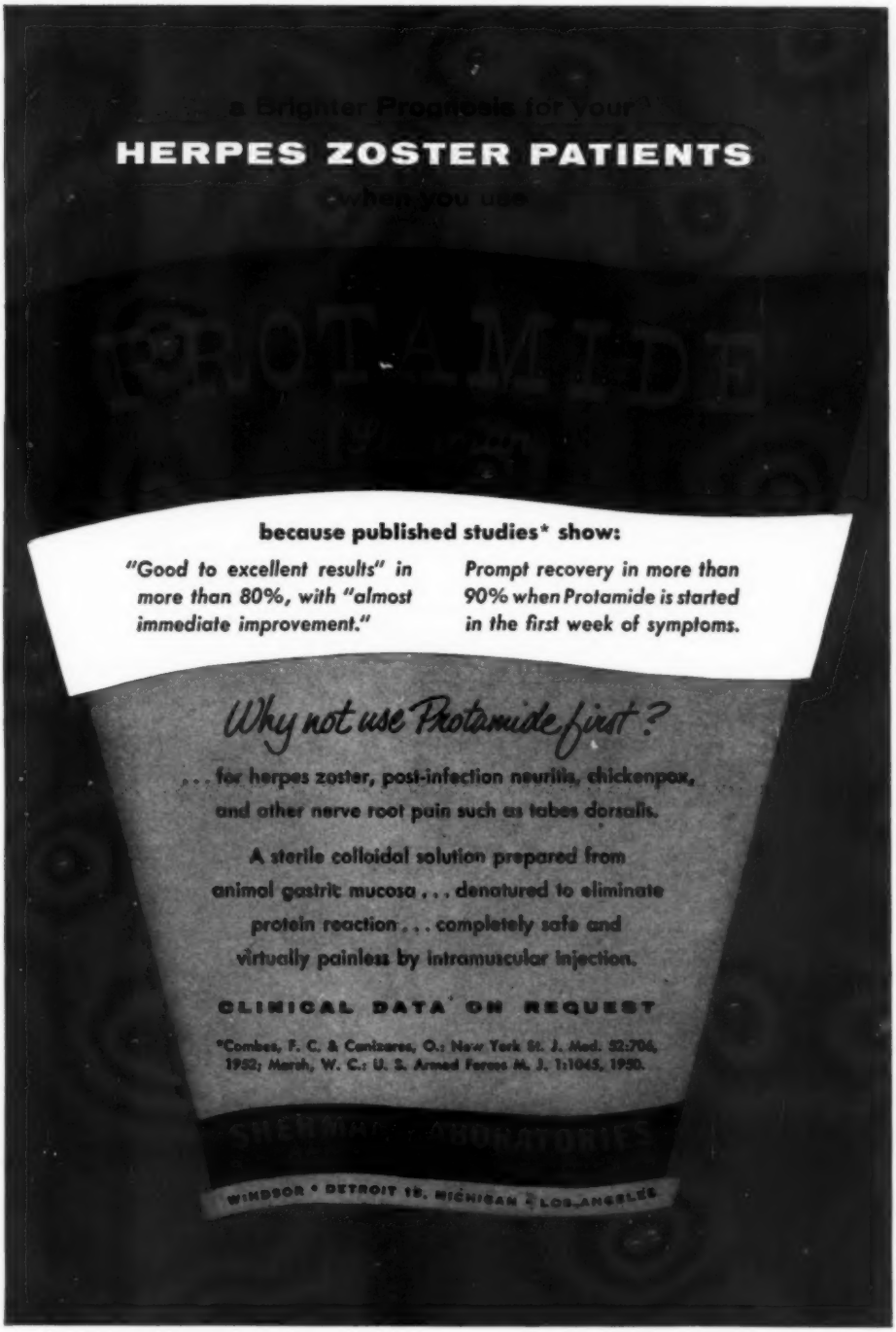
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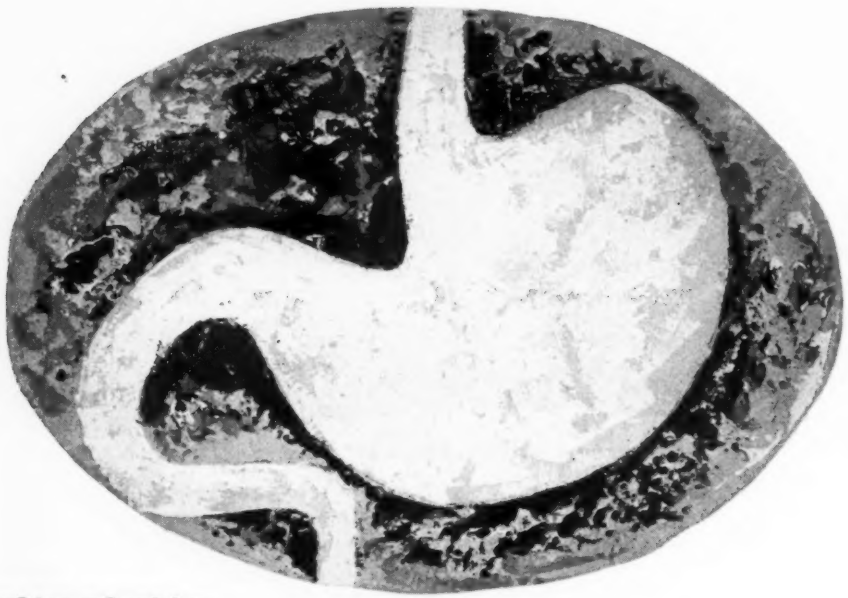

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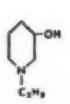

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The Journal of the American Osteopathic Association

PUBLISHED MONTHLY BY THE AMERICAN OSTEOPATHIC ASSOCIATION

212 East Ohio St., Chicago 11, Ill.

Vol 54, No. 10 June, 1955

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Muscle and the Myoneural Junction

Their Relation to Segmental Spinal Dysfunctions

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INTRODUCTION

A considerable amount of experimental evidence is available concerning the anatomy and physiology of the neuromuscular apparatus. Because of the close relationship between segmental spinal dysfunctions and the neuromuscular mechanisms it seems worth while to review the anatomy and physiology of the structures involved. Reflex activity in both somatic and visceral function is well established, and the importance of segmental spinal dysfunctions and their relationship to visceral disease is recognized clinically.

In any discussion, even though it is based to some extent upon original observation, there is danger of transferring certain experimental findings to a situation in which they are not applicable. This will be avoided wherever possible in this report. Another difficulty is that many histologic technics have been employed in the demonstration of various neurologic structures. This is because all of the structures in a single neuromuscular microscopic preparation cannot be demonstrated by a single method. When results discussed in this report are based on different methods it will be mentioned.

The major problems concerning the structural and functional aspects of the innervation of striated muscle were recently stated as follows,[1] (a) recognition of the diversity of innervation in different species of vertebrates, (b) extent to which morphologic variations in the motor endings reflect the age of the animal, (c) individuality of motor innervation in different muscles of a single animal, (d) existence of single muscle fibers commonly supplied by several endings, (e) the relationship of the motor ending to the sarcolemma (epilemmal or hypolemmal), (f) correlation between the type of nerve fiber and the motor ending, and (g) the possibility of recognizable adjustment of efferent innervation to meet the needs of increasingly perfect proprioceptive control in different groups of animals. To this might be added an evaluation of these mechanisms on the function of the spinal column, and the status of the motor unit of paravertebral muscles segmentally related to the vertebral articular strain.

MUSCLE

Types of Striated Muscle.—

Many microscopic and macroscopic features of striated muscle have been reported and evaluated; discussion of characteristics that are well established and not recently revised will be omitted from this paper. For review of the chemical composition of muscle, the reader is referred to the work of Szent-Györgyi.[2]

The appearance of striated muscle fibers stained by different histologic technics has been reported.[3] In general the cross striations, which characterize this type of muscle, are demonstrated best by phase contrast microscopy or by the use of iron hematoxylin, while the cellular elements are demonstrated by cytoplasmic and nuclear stains.[4] Another advantage in the use of phase microscopy is that the muscle is observed without fixation or staining so that the possibility of artefact formation is reduced (Figs. 1-5).

A problem that has been reconsidered recently is the status of red and pale muscle in relation to muscle function. The possibility of the existence of two types of muscle fibers in vertebrates was first reported by Stefano Lorenzini in 1683, but Ranvier first differentiated the two types histologically and suggested there might be a difference in function.[3] The red muscle was differentiated from the pale by slowness in contraction, with the onset of tetanus at slower rates of stimulation, by the large amount of sarcoplasm in the fiber, by more distinct longitudinal striations, and by greater endurance. In the higher mammals the muscle fibers tend to be of equal size, and the amount of granulation in the individual muscle fiber depends upon the nutritional status of the fiber.[5] It has even been suggested that there may be a conversion of red muscle to pale as the result of motor activity.[6]

Hay[7] reported that in general the red muscle was supplied by nerve fibers of small diameter. This would indicate that the response of red muscle fibers is slower than that of pale muscle fibers, a conclusion that would be in agreement with the criteria stated above; however, the matter is still controversial.

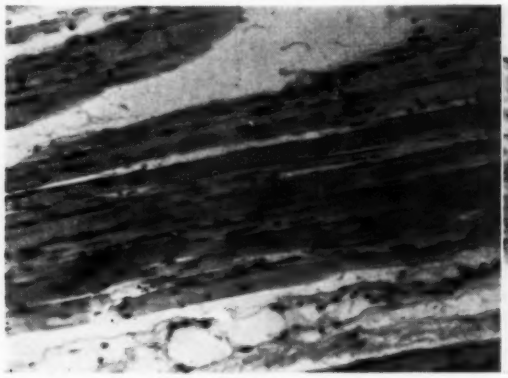

Fig. 1

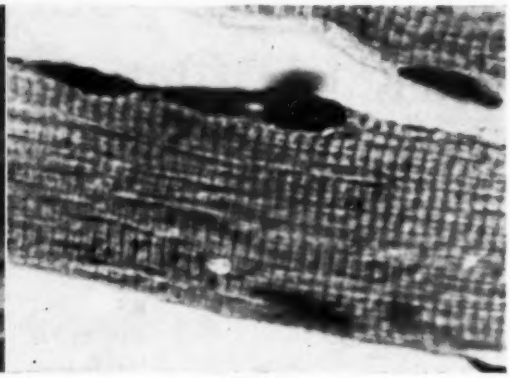

Fig. 2

Fig. 1. Striated muscle, ×100. Routine section of striated muscle stained with hematoxylin and eosin, demonstrating the even, parallel edges of the muscle fibers. Cross striations do not show clearly with this stain and at a magnification of 100. Blood vessels and connective tissue in lower middle portion of the photomicrograph. Fig. 2. Striated muscle, ×450 and enlarged. Single muscle fiber stained with iron hematoxylin and eosin. The cross striations and nuclei of the muscle fiber are demonstrated by this method.

Specific functional capacities for the two types of muscle have not been definitely established. In both repeated nerve impulses cause a summated mechanical contraction or "tetanus" in which the twitches are partially or completely fused, according to the rate of stimulus.[8,9] In red muscles completely fused contraction will result at rates of excitation as low as 30 per second and in pale muscles, 100 per second; but in the extraocular muscles as many as 300 stimuli per second are required. Such a classification would indicate that generalizations concerning muscle function may lead to erroneous conclusions, and when evaluations of the spinal muscles are contemplated the specific and precise composition of the musculature should be ascertained.

When teased fibers stained by gold chloride are examined, it first appears there is a difference between the two muscle types. On observing the entire length of the muscle fiber it is obvious that the difference is due to a variation in the impregnation and staining intensity. The use of other stains has so far failed to differentiate adequately the two types of muscle fibers.

The usefulness of the differentiation of pale and red muscle fibers in the evaluation of segmental spinal dysfunctions remains speculative. The criteria for the differentiation into two distinct groups would indicate that fibers of red muscle are primarily involved in experimental vertebral articular strains.

Contraction and Contracture.—

When considering the reactions of the paravertebral musculature in association with spinal dysfunctions it seems pertinent to differentiate between contraction and contracture. Even though the two terms have been used interchangeably each has a definite meaning, and each describes a specific physiologic state.

If a single fiber is stretched between supports to its rest length and is stimulated tetanically at several points along its length simultaneously, it will everywhere present a uniform appearance. When the supports are moved nearer to each other by an amount equal to 15 per cent of the resting length and the muscle is stimulated at one end, the moment stimulation begins that end shortens and takes up all, or most, of the slack before the end distal to the application of the stimulus begins contracting. About one third of the fiber is enormously shortened and bulged out, while the balance remains about normal in diameter. Whenever a fiber must first develop tension before over-all shortening takes place, the effect of the load is to equalize the patterns of shortening, irrespective of where the fiber was first stimulated; the load can govern the pattern of shortening.[10] These processes are considered to be contractions.

Contracture[11] has a variety of meanings and has been used to indicate different states or processes in

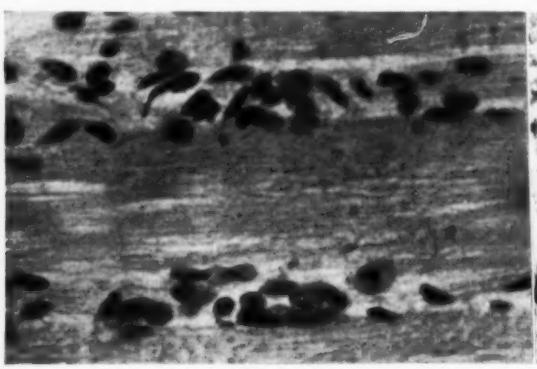

Fig. 3

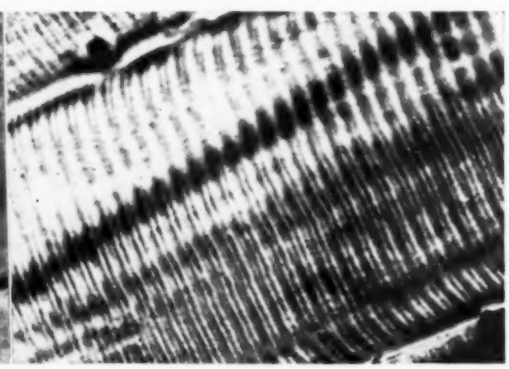

Fig. 4

Fig. 3. Striated muscle, ×450, stained with Harris' hematoxylin. Aggregations of oval hyperchromic cells near the edge of the muscle fiber. Long cells are the cells of the sarcolemma. No nerve elements are demonstrated by this method. Fig. 4. Striated muscle, ×450 and enlarged, without fixation or stain. Photographed with medium contrast phase contrast system. Nuclei of the muscle fiber are not demonstrated; cross striations are adequately demonstrated.

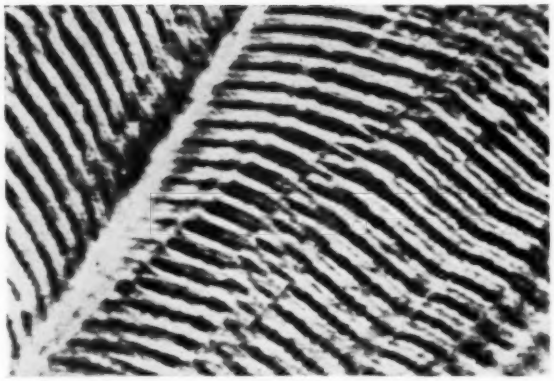

Fig. 5

Fig. 5. Striated muscle, ×450 and enlarged. Similar to Figure 4 but photographed with B minus phase contrast system.

striated muscle. It can be defined as a prolonged reversible but not propagated shortening of muscle; it can be regulated, the strength graded, and the activation stopped at will. The variable changes, being slow, can be observed under a microscope. Contractures are usually obtained by direct excitation, not through the nerve, although under certain conditions local shortenings at the neuromuscular junction may follow nerve stimulation. Contractures have been set up by constant currents and by drugs that produce the condition directly upon application.

Gasser[12] differentiated contracture from contraction by stating that in contraction repeated stimuli cause a series of twitches and when close together, a sustained contraction. This is to be contrasted with certain states of sustained shortenings which were considered to be incomplete involvements of the muscle fibers, which were not propagated and not attended by a wavelike electrical variation. The condition of contracture included the following points: (a) at least some part of the normal contractile mechanism be involved, (b) the changes must be reversible, (c) tetani are excluded, and (d) it may be produced by mechanical, electrical, or chemical stimuli.

Others[3] have compared tetanic shortenings to states in which the muscle shortens or maintains tension without evidence of repeated neural excitation of the contractile mechanism. Such shortenings accompanied by some part, but not all, of the usual evidence of activity of the contractile mechanism and reversible were designated by the term "contracture"; but once the situation became irreversible, they were designated as rigor.

An interesting point was introduced by Gilson and others[13] with a suggestion that residual contractures may be differentiated from mere prolongations of the response proper. Distinction must be made between responses which are complicated by arising within the muscle as a tissue and those arising within the muscle as an organ. From the standpoint of the evaluation of the vertebral articular strain such a differentiation seems superficial as long as certain fibers associated with the strain exhibit both histologic and physiologic variations, and there is experimental and clinical evidence that they do.

Investigations of the deepest layer of the segmental vertebral muscles associated with an experimental vertebral articular strain indicate that a situation exists that may be classified as contracture; the investigations were histologic evaluations made with the phase contrast microscope.[14] There was no evidence that suggested that either one or both types of muscle fibers were involved in the state of contracture.

THE MOTOR ENDING

Structural Characteristics of the Motor Ending.—The embryologic development of the myoneural junction (motor end plate) was described by Boeke.[15] For the most part the development of the myoneural junction is relatively unimportant in this discussion. The exception to this is the explanation of the occurrence of "double" or "accessory" endings. These are considered to be developmental variations and occur by the splitting of the epilemmal axon and the formation of a motor ending on each branch. Some of these are complete; however, some are incompletely divided, which results in a motor ending twice the size of the average for the muscle group.

The microscopic characteristics of the motor endings of an adult animal are different when different staining technics are used. Certain stains demonstrate the nerve elements and others the cellular elements, so that disagreements have developed concerning the typical structure of the motor ending. In general the protoplasm of the motor ending is an expansion of the nerve termination over the sarcoplasm, with a fine granular material in and around the plasmodesmata. The junction of the epilemmal and hypolemmal axons is sometimes slightly elevated and is termed Doyère's hillock or eminence. The cells of the motor endings (which cannot be demonstrated by the gold chloride stain) lie within the branches of the plasmodesmata. In man the motor endings are about 45 micra in length (pectoralis major) but in smaller animals, such as the rat, the average is about 30 micra. In each instance the measurement was made along the longest axis of the oval motor ending.

By staining with gold chloride the neural extensions of the efferent nerve are well demonstrated; cellular stains such as a modified Schiff's reagent are necessary to demarcate the cellular elements.

Boeke[15] described a delicate network of nerve fibers in Bielschowsky (silver) preparations, stretching from the neurofibrillar branches of the plate to make intimate contact with the myofibrils (periterminal network), and considered the whole structure to be under the sarcolemma (hypolemmal). By using a modification of Schiff's reagent similar networks have been demonstrated, but not as clearly demarcated as desirable. However, the position of the endings was used to differentiate between the motor and the sensory, as epilemmal endings were considered to be sensory. This classification has lost value because both sensory and motor endings have been shown to be epilemmal.[16] The terms "epilemmal" and "hypolemmal" should, therefore, not be used in referring to the efferent nerve and its terminal expansions.

The location of the motor endings in relation to the sarcolemma would seem to be a minor point. When considering the mechanisms of neuromuscular transmission the exact location is of importance, and more recent investigations have placed the motor ending in an epilemmal position and have also considered the possibility that it is encapsulated.[17,18]

Gold chloride staining often indicates that motor endings are enclosed in a capsulelike structure;[19] however, its demonstration has not been too satisfactory. Stains that show the cellular elements of the motor ending do not demonstrate a capsule, even though they

Fig. 6

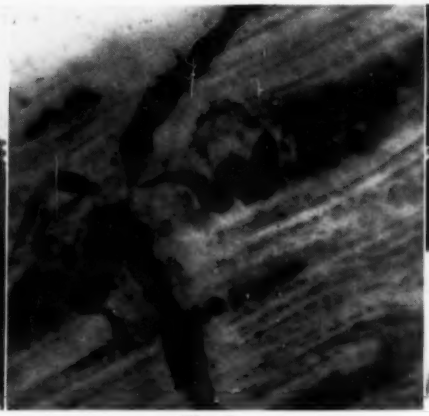

Fig. 7

Fig. 8

Fig. 6. Motor ending, ×100, stained by gold chloride. Nect urus. Relatively large motor ending with double, or split, efferent nerve. Fig. 7. Motor ending, ×450, stained by gold chloride. Frog. *Terminaison en ligne,* large area of nerve contact with the muscle. Fig. 8. Motor ending, ×450, stained by gold chloride. Lizard. *Terminaison en plaque,* oval compact ending typical of the more advanced species.

do demonstrate the other connective tissue elements. This suggests the possibility that the granular material (of Kühne) may form a "capsulelike" structure which is stained only when the neural elements are stained.

Structural Variations in the Motor Endings.—

The assumption that the motor endings in various muscles of a single species are all the same seems to be false, even though in certain muscle groups of the rat there is a striking similarity in both size and shape in the segmental paravertebral muscles when compared with the rectus femoris.[20] Preliminary investigations in the comparison of more muscle groups in a single species indicates that there is a variation in both size and shape of the motor endings, which seems to be dependent upon the functional requirements of the muscle.

It has been suggested that there is a difference in the motor endings of red and pale muscle. Observations of gold chloride stained muscles of the foot (including both extensors and flexors), deep and superficial paravertebral muscles, and the tongue of the rat do not support such a suggestion. However, it is stated[21] that in the rat the extensors, which are classified as pale muscle, receive larger nerve fibers than do their flexor antagonists and that many efferent fibers to pale muscles are larger than those to red muscles when both types are present in the same muscle.

The motor endings of certain rat muscles exhibit morphologic variations when compared with those of other groups; the diaphragm does not seem to have characteristic endings, and the variations occur consistently enough to be considered typical rather than artefacts. No differentiation of the red and pale muscles could be attempted on the basis of differences in staining or in the shape or size of the motor endings.

The adaptation of structure to function of the motor endings is not limited to mammals. Throughout the subphylum vertebrata such adaptations have been observed.[22,23] These differences seem to be biochemical as well as morphologic because alterations in the pH of the solutions have been found necessary to demonstrate the motor endings properly.[24]

Various classifications of motor endings have been attempted, both from the standpoint of the shape of the ending and the size of the muscle fiber.[25] It has been stated that *terminaisons en grappes* were observed in muscle fibers with a diameter of 10 to 60 micra and that *terminaisons en plaque* were observed on fibers with a diameter of 90 to 150 micra. More recent investigations fail to support such conclusions; it is unusual to observe muscle fibers with varying widths in a single muscle and in some muscle groups, as the diaphragm. In the latter both endings occur. A classification based on the diameter of the muscle fiber will be misleading.

It seems well established that three types of motor endings do occur in the subphylum vertebrata, although no one species seems to have all three. The motor endings are classified as *terminaisons en plaque, terminaisons en ligne,* and *terminaisons en grappes.* The first is the characteristic ending in the mammal, although in some muscle groups such as the diaphragm *terminaisons en grappes* are present. The *terminaisons en ligne* occur chiefly in amphibians although they were also observed in reptiles. In a review Hinsey[16] mentioned that ultraterminal endings, which consisted of unmyelinated branches from the *terminaisons en plaque* ending on the same or adjacent muscle fibers, did exist. The gold chloride method does not demonstrate such an arrangement; however, the method stains unmyelinated fibers poorly and no conclusions should be drawn.

That functional adaptations are possible in the shape of the motor endings was suggested by Tiegs.[3] He stated that the presence of very numerous minute motor units of the eye muscle is customarily associated with a high degree of movement. Of the unique character of these muscles there is no doubt; they are outstanding in the consistency and delicacy of movement and in the extreme precision of their postural mechanism. It is significant that with large motor units adaptation probably does not occur, at least to the same degree. A similar situation does seem to be present in the muscles of the tongue.

The variation of motor innervation in different species of vertebrates has been mentioned, and in spite of the fact that a detailed investigation of all the vertebrates has not been done, sufficient evidence is available to indicate the major differences among species. The most significant observation has been that morphologic adaptations occur throughout the subphylum vertebrata, indicating that a high degree of functional adaptation is present. In certain animals (amphibians) where sudden intensive motor responses are required, the motor

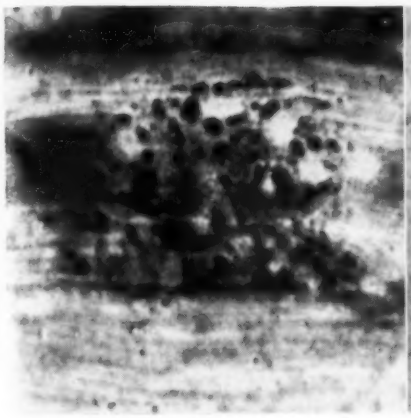

Fig. 9

Fig. 10

Fig. 11

Fig. 9. Motor ending, ×450, stained by gold chloride. Bird. Ending classified as *terminaison en plaque* but does not exhibit the compactness characteristic of the higher vertebrates. Fig. 10. Motor ending, ×450, stained by gold chloride. Rabbit. Compact ending typical of the rabbit. Fig. 11. Motor ending, ×450, stained by gold chloride. Rat. "Doubling" of the motor ending. Efferent nerve divides near the termination which eliminates the possibility of a plurisegmental origin. "Capsulelike" structure around the motor plates.

endings are elongated so that the nerve is in contact with the muscle for some distance. This arrangement is modified in the reptile and in some species (snake) definite *terminaisons en plaque* are present. In the birds the characteristic ending is a *terminaison en plaque,* without the compactness that is characteristic of the mammalian ending; in the flying birds a very loose ending is characteristic permitting a larger surface of nerve-muscle contact.

The evaluation of the motor end plates in a series of vertebrates and of muscles of a single species seems to answer the question concerning the possibility of recognizable adaptation designed to meet the needs of increasingly complicated control in different groups of animals. There are recognizable differences between classes of the subphylum vertebrata when two groups of muscles are examined, the rectus femoris (or its homologue when present) and the paravertebral musculature (when present). Within the class the motor endings on these two muscle groups show a great degree of consistency. When more muscle groups of a single species are examined there are differences in both the size and morphology of the motor endings.

It is probable that motor endings reflect the age of the animal to some extent, especially in the very young. In a series of rats in which comparisons were made of the motor endings of different muscles it was observed that there were more inconsistencies in appearance and morphologic characteristics in the rats weighing less than 100 grams than in those weighing more than 150 grams. In the smaller rats muscle coordination and activity seemed normal; therefore, it could not be suggested that an adequate nerve supply was not present. The only deduction that can be made from this evidence is that the motor endings undergo a period of maturing and that the morphologic and biochemical characteristics do not become consistent until maturity.

The next question concerned the possibility of individual characteristics for motor endings in different muscle groups. In reviewing histologic preparations from a series of adult rats, including the tongue, intercostals, paravertebrals, rectus femoris, gastrocnemius, and muscles of the foot and diaphragm, some variations were noted. The motor endings in the tongue and foot are much smaller and more compact than those of the other muscle groups examined, and in these two groups the motor endings are similar. In the intercostals, rectus femoris, and gastrocnemius there is some "doubling" and the occurrence of loose motor endings. The loose ending, or *terminaison en grappes,* is found in the diaphragm for the most part. As these observations were made on gold chloride stained sections there was the possibility that incomplete impregnation of the tissue by the stain might have resulted in artefacts; only sections with a similar staining intensity of the muscle fibers were compared (Figs. 6-16).

Physiologic Variations in Motor Endings.—

Carey[26] suggested that the morphology of the axonal termination in the motor end plate reflects the functional state of the motor unit, and there are variations in the morphologic characteristics of the motor endings in muscles under different conditions. A more recent report states[3] that it is doubtful if the above statements are significant. When considering the adaptations of the motor endings on muscles that subserve specific functions it does seem possible that a certain amount of structural adaptation might occur, although there seems to be no reason to expect variations in a single motor ending under slightly different circumstances.

Carey[27,28] also reported that a number of experimental conditions caused morphologic alterations in motor endings. Starvation in the albino rat, fatal within 8 to 15 days, produced a structural detachment of about 20 per cent of the motor endings in skeletal muscle, morphologically manifested by progressive retraction and eventual uprooting of the end plate in the atrophic muscle fiber and loss of the granular sole plate. In other experiments using either electrical stimulation, curare, or lactic acid, similar alterations were observed which also included an enormous distention of the efferent nerve resulting in structural alterations of the motor end plate. In other experiments and following stimulation to the efferent nerve a type of migration, termed ameboid motion, was reported.

Other investigators have disagreed with the above conclusions. It has been stated that no ameboid motion occurs in the motor endings, but that they move passively with muscle contraction; this reaction was observed in invertebrates by phase contrast microscopy.[29]

Fig. 12

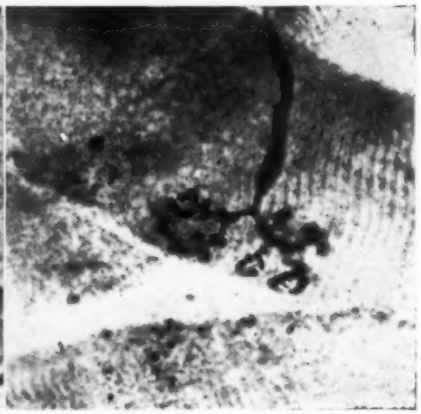

Fig. 13

Fig. 12. Motor ending, ×450, stained by gold chloride. Rat. Motor ending from muscle of the foot. Small, compact ending typical of these muscle groups. Fig. 13. Motor ending, ×450, stained by gold chloride. Rat. Motor ending from gastrocnemius of same animal. Note the differences in the size (motor endings in Figures 12 and 13 taken at exactly the same magnification.)

Experiments using electrical stimulation have failed to obtain such a response but did suggest that staining variations were present in the experimental sections.[30,31] Dublin[32] did not obtain a similar response of the motor endings from the application of curare or hypothermia; however, there was an expansion of the motor end plate following exercise. Another report[33] suggested that there was an alteration of the staining reaction following the production of an experimental error in locomotion and electrical stimulation. In the case of the former there seemed to be a high degree of adaptation to the stress, and if the animal was permitted to live for several weeks no staining variation was noted. Such evaluations must be carefully controlled due to the capricious activity of the gold chloride impregnation technic.

Multiple Innervation of Muscle Fibers.—

The problem of multiple motor endings on a single muscle fiber is still unsettled. Recently it was stated that in a well-stained preparation of a small muscle it is clear that each muscle fiber receives only one efferent nerve and has but one motor ending.[3] In a small muscle fiber such as one from the foot of the rat, this seems to be true. Other investigators have reported that more than one motor ending has been observed on a single muscle fiber; whether or not these were from the same spinal cord segment was not stated. Hodes[34] concluded that some experiments seem to present both histologic and physiologic evidence for the innervation of a single muscle fiber by two motor nerves. Although data are scanty on this point, innervation of a single muscle fiber by two motor nerves must be considered likely in view of the more complex histologic studies of others[35-37] (Fig. 11).

Hinsey[38] stated that somatic fibers branch so that each anterior horn cell supplies a number of muscle fibers. Tiegs[1] concluded that each muscle has at least one motor ending but did not exclude the possibility of double motor innervation. It was also stated that as high as 125 muscle fibers may be supplied by one efferent nerve through repeated divisions of the peripheral supply. Recently[39] a report stated that even though several groups of anterior horn cells were degenerated the overlapping of the peripheral units permitted a certain amount of adaptation to occur in spite of a degree of cord damage.

It is certain, then, that some if not all muscle fibers are supplied with two motor endings. The question still to be answered is whether the fibers originate in the same or different spinal cord levels.

Cells of the Motor Endings.—

The cellular characteristics of the motor endings

Fig. 14

Fig. 15

Fig. 16

Figs. 14-16. Motor endings, ×450, stained by gold chloride. Rat. Motor endings from the diaphragm of the same rat. Variation in size and arrangement of the terminal nerve net.

Fig. 17

Fig. 18

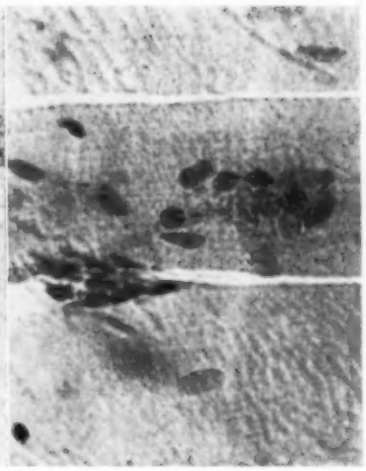

Fig. 19

Figs. 17-19. Motor endings, ×450, stained by a modification of Schiff's reagent. Muscle from gastrocnemius of rat. Efferent nerve can be seen in Figures 17 and 18. Cells large and somewhat granular. Figure 19 demonstrates the aggregation of cells on surface view.

were first described by Boeke[15] using silver staining methods, and the relationships of the terminal nerve branches and the cells of the sole plate (motor ending) were described. The difficulty in demonstrating these relationships by other technics has been mentioned, for the gold chloride method stains only the nerve, and possibly only myelinated, elements.

In the examination of a series of preparations stained with iron hematoxylin and a modification of Schiff's reagent, it became obvious that there are aggregations of nuclei in association with the muscle fiber that are not similar to the nuclei of the sarcolemma as demonstrated by the use of Harris hematoxylin and eosin, although the latter were also stained. The cells of these aggregations were large and oval, with a slightly granular cytoplasm and a small hyperchromic nucleus. In some preparations (teased) the efferent axon was observed, and it terminated in relationship to these clumps of cells. The final disposition of the terminal twigs of the axon could not be seen.

It was concluded that these cellular aggregates were the motor endings; the arrangement of the cells and as much of the nerve as could be seen in no way differed from the observations of Boeke (Figs. 17-19).

SENSORY ENDINGS

Types of Sensory Endings.—

It appears that the sensory endings of striated muscle and of the tendinous insertions of the striated muscle have received less attention than the motor endings, even though they are essential for reflex activation of the motor endings.

Kühne[40] first described the muscle spindle, although it apparently was Sherrington[41] who first recognized it as a sensory organ of muscle. Hines and Tower[42] described the innervation of skeletal muscle in detail and included the sensory as well as the motor innervation.

Two endings, one enclosing the intrafusal muscle fiber and the other in the tendon, constitute the sensory receptors of skeletal muscle. The annulospiral organ (muscle spindle) is considered to transmit stretch re-

Fig. 20

Fig. 21

Fig. 22

Figs. 20 and 21. Sensory endings. Figure 20, ×100; Figure 21, ×450. Rat. Organ of Golgi located near the termination of the muscle fiber. Diffuse distribution of the terminal nerve endings. Fig. 22. Sensory ending, ×100, stained by gold chloride. Rabbit. Organ of Golgi. Distribution of the nerve endings between the connective tissue cells of the tendon.

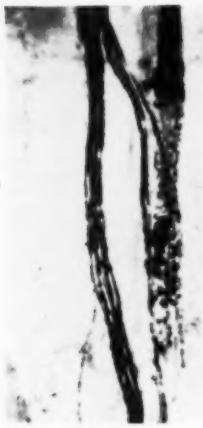

Fig. 23

Fig. 24

Fig. 25

Fig. 26

Fig. 27

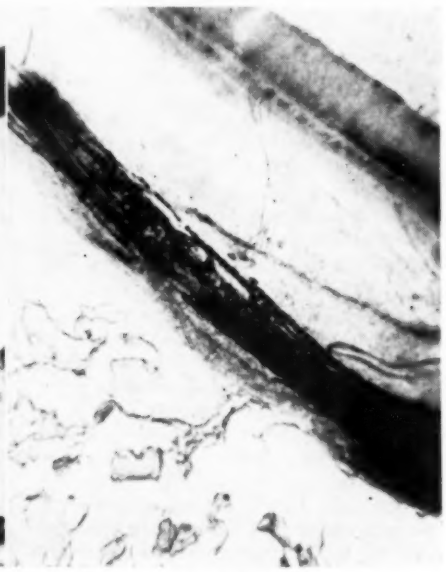

Fig. 28

Figs. 23-28. Annulospiral endings, ×100, stained by gold chloride. Figures 23 and 24, hamster. Figures 25 and 26, rat. Figure 27, monkey. Figure 28, human. Diffuse pattern of the nerve around the muscle fiber makes it difficult to distinguish between the species.

flexes; while the tendon receptor (organ of Golgi) is believed to transmit impulses originating from tension on the tendon, stimulation (activation) of this receptor leads to reflex inhibition.[43] Whether or not pain is mediated by this system is open to question. In this regard Hinsey[16] reported that there is increasing evidence that pain is mediated by small nerve fibers, some myelinated and some unmyelinated, originating in the dorsal root ganglion and having a skeletal distribution, some from the blood vessels and some terminations near the muscle fiber.

Ruffini[43] in the study of muscles of the cat distinguished two distinct types of sensory endings and considered the annulospiral bundle as the primary ending and a second type, the flower-spray, as secondary. In a series of mammals, including the hamster, kangaroo rat, mouse, cat, rat, and rabbit, the scarcity of the flower-spray ending suggested that it should not be considered as a typical sensory receptor. Prosser[44] concluded there is no evidence that correlates the structure of the sensory endings to a specific function. However, in considering the consistent and specific location of the organ of Golgi and the annulospiral bundle, it appears that an exception should be made in these instances.

Structure and Function of Sensory Endings.—

All of the annulospiral endings in any one capsule are derived from a single large myelinated fiber; in many cases this divides into several branches before innervating the muscle fiber. This is mentioned because Ruffini[43] concluded that the flower-spray ending differed in that each ending had a separate nerve fiber and consisted of a branched network which enfolded the myotube between the polar and equatorial part of the intrafusal fiber. The existence of encapsulation of the annulospiral organ has been questioned, but there seems to be greater agreement that it does than in the case of the motor ending.

In muscles stained by gold chloride the morphologic characteristics of the annulospiral ending are

similar in species of the subphylum vertebrata; however, no endings of this type were observed in fish. In this series the flower-spray ending was extremely rare, so much so that its occurrence was not considered significant. Annulospiral endings, although consistent in occurrence, were not as numerous as motor endings.

Concerning the neurotendinous endings Matthews[45] described thin encapsulated specialized endings, each of which surrounded a bundle of tendinous fibrils which received attachments of the muscle fibers. They were situated where the muscle fibers joined the aponeurosis and were not actually in the tendon. Each of these organs gave rise to a single nerve fiber and was so placed as to be responsive to tension on the attached muscle group; it was found that the threshold to the tension was high. The tension produced by active contraction was much more effective than that produced by passive stretch (Figs. 20 and 21).

In some teased preparations stained by the gold chloride method such endings seemed to be present; however, the most common type of tendinous ending was that classified as the organ of Golgi (Fig. 22). There was no indication that either one of the tendon receptors was encapsulated.

There is little reported concerning a differentiation between reflexes emanating from activation of the annulospiral endings in comparison to those from the organ of Golgi, except the fact that the former are stretch and the latter tension reflex organs. Brookhart[46] stressed that reflex activities accompanying excitation of the annulospiral ending must apparently be differentiated carefully from reflex activities initiated by excitation of the tendon organs on the basis that tendon organ activation inhibits motor neurons to the same and synergistic muscles and facilitates the response to the antagonists. This should be considered when attempting an evaluation of the response of the segmental paravertebral musculature in association with the vertebral articular strain; in spite of the presence of facilitation in spinal reflex activity, it appears that there may be certain inhibitory reflexes activated.

The morphologic characteristics of the annulospiral receptor and the organ of Golgi did not vary in experimental animals with an error in locomotion or following electrical stimulation, even though a change in the staining reaction of the motor ending had been obtained in the same animals, and there was a great degree of consistency of structural characteristics in different animals (Figs. 23-28).

It is interesting to note that a recent report[47] stated that the annulospiral endings are peculiarly resistant to muscular disease and that the ending and its muscle fiber are therefore more prominent in sections of atrophic muscles, whether from neuronal atrophy or from muscular dystrophy, even after all of the surrounding muscle fibers have been reduced to thin sarcoplasmic threads.

Function of Sensory Organs in Relation to Spinal Strains.—

An interesting point concerning the functional capacity of the sensory elements as compared to the motor endings is that the sensory organs of striated muscle do not fatigue readily.[48] The significance of this is that in the presence of the vertebral articular strain sensory impulses may continue to pass to the spinal cord in the presence of reduction of motor activity. Therefore, when the state of contracture is present in the spinal musculature the sensory elements are activated, and this may persist for indefinite periods and become an irritable focus.

To the osteopathic physician the problem of neuromuscular response is of primary concern because it is chiefly through such a mechanism that segmental spinal dysfunctions have a potential for influencing visceral function. The histopathologic effects in segmentally related viscera indicate that the primary visceral response is in the vascular supply (controlled by autonomic nerves), while the secondary effects of the disturbance in the blood supply may be noted in the cells of the organs themselves.[49] Several explanations have been offered that suggest the importance of the nervous system, and the involvement of striated muscle and sensory and motor units in areas of segmental spinal dysfunction.[50,51] These include the fact that segmental striated muscle groups become an irritable focus; sensory and motor unit activity then becomes part of this reflex mechanism. Histologic examination of the anterior horn cells, motor endings, and striated muscle of the involved segment demonstrates that there are alterations in these structures; however, they are not considered to be irreversible.[52]

The clinical observation that "chronic" vertebral articular strains are not extremely painful might be explained on the basis that sensation may not accrue although a central reaction may be established. In many cases it is hazardous, and in many immaterial, to state whether sensations do or do not attend the central reaction.[53] This evaluation of the reflex patterns indicates that the involuntary reflex has a potential for affecting the organism, even though conscious perception of the dysfunction has not taken place.

There seems to be no disagreement with the opinion that the primary reaction to the vertebral articular strain is a reflex mechanism. The fact that the osteopathic physician regards these reflexes as important basic reactions does not mean that they have not been recognized by others. Tower and Bodian[54] confirmed the conclusion reached after a study of involved segments in dogs that mammalian cord mechanisms operate only under the stimulus of arriving nerve impulses. Deprived of such excitation the cord produces no activity which reaches effectiveness in the skeletal musculature. Such an explanation seems to be applicable also to the concept that vertebral articular strains can produce a reaction in the striated musculature and that the initial reflex reaction does perpetuate itself for indefinite periods, involving the sensory and motor units of the organism.

SUMMARY

The problem concerning the existence of red and pale muscles in mammals was reviewed, and it was pointed out that differentiation by the use of a modified Schiff's reagent, gold chloride, and hematoxylin and eosin has not been achieved.

The possibility that motor endings on red and pale muscle fibers are morphologically different could not be demonstrated by the gold chloride staining method, a technic that results in adequate differentiation of the neural elements.

The cellular elements of the motor ending were demonstrated by the use of a modified Schiff's reagent, although the results were not completely satisfactory; the final termination of the efferent nerve was demonstrated by the method.

The concept of contracture in striated muscle was reviewed and the similarities between the descriptions

of this state and the muscles associated with vertebral articular strains were indicated.

The variation of motor endings in the vertebrates was described, and the fact that in some muscle groups of a single species there are morphologic variations in the motor ending was indicated.

The evidence for double motor endings on a single muscle fiber was reviewed; the lack of evidence that such an innervation was intersegmental was also mentioned.

The evidence against extreme morphologic variations occurring in the motor endings under extreme pathologic conditions was reviewed; it was suggested that the motor endings move passively on motion of the underlying muscle fiber.

The lack of morphologic variations in the annulospiral organ and the organ of Golgi in a series of vertebrates was mentioned, and also the lack of morphologic variations in appearance under experimental conditions was indicated. The functional significance of the two endings was reviewed.

A correlation was made between the motor, sensory, and muscular elements in association with the vertebral articular strain.

Acknowledgments

The original investigations mentioned in this report were supported by a grant from the Bureau of Research of the American Osteopathic Association. The author wishes to thank C. C. Hassett, Ph.D., for his criticism and advice in the preparation of the paper.

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Suffocation or Sudden Death in the Infant

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INTRODUCTION

One of the most disconcerting problems of pediatric pathology is the sudden and unexpected death of an apparently healthy infant. Sudden death is always a shocking event, and parents are filled with remorse and self-accusation when they find their child dead a few hours after he has been put into his bed, especially when the child is found under the bed clothes. Immediately the parents think of "smothering" and blame themselves for neglect.

There has been no plausible explanation for these sudden deaths, and the subject is a ripe field for inquiry. Up to the present the lack of serious and concerted consideration has been partly due to the infrequency with which the physician and hospital are called upon to deal with this problem, as these deaths fall in the realm of the local police department and the office of the medical examiner. However, autopsies are not carried out in all cases, and the term "sudden death due to suffocation" is used in many instances where it is erroneous. The term should not be used unless there is clear physical evidence of strangulation.

Ben H. Brown,[1] Coroner of Los Angeles County, states, "We do not use suffocation, strangulation or asphyxiation upon a death certificate without an explanation of the motivating circumstances. We never use the words 'sudden death' upon any certificate." Carroll[2] has said, ". . . many of the diagnoses set forth on the death certificates were placed there to satisfy the Bureau of Vital Statistics."

Recently pathologists have become vitally interested in the problem and have stated that the more complete the history and autopsy are, the less likelihood there is that a sudden death will be listed on the certificate as one due to accidental suffocation, status thymicolymphaticus, or aspiration of stomach contents. Gross necropsy findings often are insufficient to explain death, but more critical histopathologic examinations of the tissues have yielded significant causes in these unexplained cases and are providing clues for further studies.

A plea has been made for more thorough autopsies of all organs, including the middle ear, mastoid, larynx, vocal cords, hilar areas of the lung, and several areas of the brain. In addition bacterial and viral cultural studies should be carried out, as well as blood cultures, complete blood count, and blood sugar examinations; and, most important, the level of gamma globulin found in the infant should be determined as soon after death as possible. Adelson[3] has also suggested that an epidemiologist visit the home of the infant and thoroughly investigate all details of the infant's remote and recent past, and that a detailed family history be taken.

PRIMARY CAUSES OF SUDDEN DEATHS

What is the usual picture of these sudden deaths of infants?

1. An apparently healthy infant, with no perceptible signs of illness, is put to bed and a few hours later is found dead.

2. The majority of these sudden deaths occur in infants from 1 to 6 months of age, with the peak at 2 to 3 months.

3. The seasonal incidence seems the greatest during the fall and early winter months, although some deaths occur during the spring months when respiratory infections are most prevalent.

4. If no autopsy is performed, the case is usually certified as death from accidental suffocation. However, the majority of the cases that come to autopsy show infection of the respiratory tract as being responsible for the death. Statistics list respiratory deaths as exceeding other causes of sudden death in infants. The most obvious reason that these deaths have heretofore been accepted as due to suffocation is because most of the infants have been found under the bed covers.

Morhardt, author of the book, "Death-Mechanisms, Causes and Certification,"[4] has written as follows:

> It is reasonable to be mystified why these youngsters are found under the covers. I am convinced that this universal tendency in such cases is not a response to asphyxia for such would serve no purpose. The fetus and infant pass through developmental phases in which they reflect both mentally and physically the various stages of animal life through which we, as humans, have metamorphosed. I feel that the reaction of infants under such circumstances reflects only this instinctive impulse to hide away while gravely ill.[5]

Since the most consistent finding at autopsy in the majority of the reported sudden deaths in infants has been pneumonia or a respiratory infection of some type, this discussion will give particular attention to this group of cases. Sudden death due to congenital anomalies and other unusual organic diseases previously unsuspected will be excluded. However, there still remains a group of cases in which no infectious cause or other explainable anatomic cause of death can be determined.

My interest in this problem has been stimulated by personal tragedy. One morning we found our baby girl, 3 months old, dead under the covers. To our knowledge she had never been ill a moment, aside from feeding problems, and she was as healthy looking as a baby could be. She had no cough, fever, cyanosis, or respiratory difficulties. Since she had not been ill nor having medical attention, her death became a coroner's case. It was then we learned that in this area "suffocation" is not accepted as a cause of death in infants. Autopsy diagnosis of the cause of death was primary interstitial pneumonia.

The day after the baby's death, our other child, aged 5, came down with a severe respiratory infection; a day or so later my wife was stricken with the same disease, evidently viral in nature. The day of the baby's death the older child had been coughing, but we had felt it was her usual allergic cough.

In a discussion of sudden death in infants, Morhardt[5] states, ". . . this type of problem represents one of a fulminating and over-whelming viral or bacterial infection." He points out emphatically that:

> . . . pathologic change of the organs affected is only a measure of the volume of reaction to disease. The relative absence of gross and microscopic pathologic change in these cases bespeaks the evident lack of capacity to combat such infections on the part of infants. The lack of pathologic alteration may not be regarded as an index of the mildness of the infection. . . . The apparent lack of illness or symptoms which ushers

in this catastrophe is further evidence of the organism's inability to react to the overwhelming infection, since such criteria as fever are manifestations of defense only.

If infection of the respiratory tract is the etiologic factor, or the primary cause of death, what are the major contributing causes or the so-called X-factors? The avenues of approach to this problem are manifold, and once we can determine the true underlying cause or causes, a more positive approach can then be made to protect these infants from a tragic sudden death.

INCIDENCE

Just how common or prevalent is this so-called suffocation or sudden death or acute respiratory death in infants? To find out, various Boards of Health in my general area were asked for a breakdown of their figures on fatalities due to respiratory diseases, and for contrast those due to common contagions, in infants under 1 year of age over a 12-month span. If the figures supplied were multiplied, in proportion to size, by the hundreds of cities throughout the country, the result would be an almost unbelievable figure.

The statistics are as follows:

City of Long Beach Department of Public Health:

Diseases	Deaths
Whooping cough	0
Diphtheria	0
Tetanus	0
Smallpox	0
Primary atypical pneumonia	7 (2 under 4 weeks of age)

Primary atypical pneumonia is the classification in the code which includes primary interstitial pneumonia.

City of Pasadena Health Department:

Diseases	Deaths
Whooping cough	0
Diphtheria	0
Tetanus	0
Smallpox	0
Primary interstitial pneumonia	6
Bronchopneumonia	4
Acute upper respiratory tract infections	1

The six who died of primary interstitial pneumonia were 1, 2, 3, 4, 5, and 8 months of age.

City of Los Angeles Department of Health:

Diseases	Deaths
Whooping cough	2
Diphtheria	0
Tetanus	0
Smallpox	0
Primary interstitial pneumonia	32 (all over 4 weeks)
Pneumonia of the newborn	15

Los Angeles County Health Department:

Diseases	Deaths
Whooping cough	1
Tetanus	1
Diphtheria	0
Smallpox	0
Primary atypical pneumonia (includes primary interstitial pneumonia and pneumonitis)	49
Pneumonia of the newborn (no mention of prematurity)	18
Pneumonia of the newborn (with mention of prematurity)	13
Infectious bronchopneumonia	28
Bronchopneumonia	6
Lobar pneumonia	4
Acute upper respiratory infection	9

ABSENCE OF CLINICAL SIGNS

What is the reason for the absence of significant clinical symptoms? Possibly it is because the infant cannot indicate that he has pain, malaise, or other symptoms and because we are dependent on observation of external manifestations of illness by the parent, physician, or attending nurse. Further, there seems to be general agreement that the body reserves of the infant are much more limited than those of the adult and that the clinical course of a pulmonary infection in the infants is insidious and rapid, producing few or no outward discernible signs. I feel that the infant is overwhelmed and goes into shock. James M. Watson[6] states:

> The constitution of an infant of three months is inadequate to take care of the overwhelming infection which is a bacteremia as well as a pneumonitis. There are no premonitory symptoms. The death in my opinion was due to toxic shock.

Holt[7] stated:

> In infants a fatal infection may run its course without producing noteworthy symptoms, even without fever. Sometimes the diagnosis can be established only by blood cultures; at other times characteristic lesions are found at autopsy, often only on microscopic examination.

Then again, although an infant may die suddenly, he may have been suffering for several days from a mild illness which was not recognized either by the parent or physician. Some studies have brought to light, after interrogation of the parents after the death of the child, that the infant was a little fussy or had a slight nasal discharge or a mild cough. Now that postmortem studies have shown that respiratory infections have been the primary cause of death, these seemingly mild symptoms take on significance.

Adams[8] reported the cases of three infants who had died suddenly without previous evidence of illness except for a slight nasal discharge or cough. At autopsy the only significant pathologic change was a marked interstitial mononuclear pneumonia associated with diffuse pulmonary hemorrhages and edema. The author suggested a virus etiology because the marked mononuclear infiltrations demonstrated in the microscopic sections were similar to those observed in experimentally produced, as well as spontaneously occurring, virus pneumonitis.

Bowden and French[9] reported the postmortem findings in forty-three children who died suddenly. In twenty-nine cases, a carefully taken history revealed evidence of several days or weeks of minor ill health.

Jacob Werne[10] has stated that he has performed or witnessed autopsies on several thousand babies over a period of 22 years without finding a single case of suffocation. He further stated that some babies seem to have an unusual susceptibility to infection, and that examinations by skilled pathologists show that most of the supposed suffocation deaths result from this cause.

In twenty-two cases of sudden deaths, Bowden and French[9] carried out systematic cultural studies which also included attempts to isolate influenza virus from respiratory tissues. In only two was the influenza virus isolated and in twenty cases histologic evidence of respiratory tract infection was found but the etiologic agent was not isolated. They state: "The seasonal incidence of these unexpected deaths suggests that respiratory infection (or some factor correlated with the incidence of respiratory infection) is an important causative agent."

Carroll[2] reported on thirty-two cases and the gross and microscopic findings at autopsy indicated some type of unknown process, but no organism could be satis-

factorily isolated. He said: "The reactions in the tissue are much too varied and nonspecific, and I feel that other factors must be involved as an underlying mechanism."

CAUSATIVE FACTORS

Is a constitutional factor involved? Carroll[2] made a postmortem study of thirty-two infants dying suddenly whose deaths were attributed to interstitial pneumonitis. He noted that these infants appeared to be slightly larger and weighed slightly more than the average child of the same age group. He noted:

> One of the clinicians . . . ventured the opinion that, although he felt these children were larger than normal, the fat was slightly softer, . . . and the child . . . did not have the usual firm feel to the tissues that other children of the same size have. . . . It is impossible to say how much significance can be attached to it. It might, however, lead one to suspect a *constitutional factor.*

Does the explanation reside in the lymphatic or vascular system? Werne and Garrow[11] in 1953 compared thirty-one unselected consecutive cases of sudden deaths in infancy with a group of violent deaths in infants. In the latter group the local lesions in the respiratory tract were not constant and when present were mild. They stated further:

> An equally significant difference was found in these 2 series of cases with reference to systemic changes in the blood vessels and the lymphatic system. . . . In not one of the examples of violent death was there loss of the normal lymph-follicular pattern or the extent of depletion of lymphocytes or hyperplasia seen in the infants found dead. In no case of death immediately after trauma was there found the same degree of hyperemia or hemorrhage in the thymus, bronchopulmonary and cervical lymph nodes. Also absent from these cases was the extreme visceral congestion. Thrombosis, which was found in 20 of the 31 cases on routine section, was never seen in those dying of trauma.
>
> The vascular changes and lymphatic tissue reactions are, in a sense, non-specific. . . . In the past 20 years we have studied also many cases of infant death reported to the Medical Examiner's Office because death occurred after a fulminating clinical illness lasting from a few minutes to a few hours. The microscopic changes were analogous to those encountered in infants found dead. The same vascular changes described in infants found dead were often prominent.
>
> One is tempted to suggest therefore, that the vascular changes common to all groups of infants under discussion may represent an important, perhaps not less important, factor in the pathogenesis of the fatal disease than the local lesion which is ordinarily accepted as a conventional cause of death.

Striking differences were also found in the lungs of the two groups. The babies who had died mysteriously and suddenly in their cribs all showed evidence of acute respiratory infection. In another group of 167 allegedly smothered babies who were studied by Werne and Garrow,[12] there was evidence in *every one* of meaningful disease, usually respiratory in nature.

IMMUNOGENIC DEFENSES

What are the immunogenic defenses of the infant to infections? Why can some infants be exposed to respiratory infections and not get them, while others do? Why do some of those who develop respiratory infections, even though they may be small, react favorably with a temperature, cough, et cetera, so that the infection can be diagnosed and treated, while others react unfavorably, go into shock, and die?

I believe the answer is in the baby itself. Those who respond to respiratory infections in a favorable manner would seem to have antibody response. Why do others not have it? Adelson,[3] Watson,[6] Morhardt,[5] and others state that the immunogenic defenses of these infants are inadequate to take care of bacterial and viral infections. Since the peak incidence of sudden deaths is at 2 to 3 months, Adelson concluded that a child is most apt to die in this abrupt fashion *after* passive immunity has worn off and *before* active immunity has been developed.

Holt[7] writes:

> To many infections, particularly those due to pyogenic bacteria, the newborn child appears to be unusually susceptible, whereas to others such as measles he generally possesses an immunity which, though temporary, is for the time being effective. His increased susceptibility is attributed to the fact that the mechanisms of developing active immunity are immature; what protection he has against infections is due largely to passive immunity from his mother, although an additional component which has been termed "natural immunity" seems to play a part.

Nelson[13] states:

> The infant does have passive immunity to some degree during the first four to six months of life for measles, diphtheria, scarlet fever and poliomyelitis, but not for . . . pneumococcic, staphylococcic, streptococcic, or influenzal infections, and apparently not for many of the viral ones.

Asher A. White[14] recently wrote:

> It is known that allergic phenomena are antigen-antibody reactions. The way in which they differ from the parent phenomena of immunity and anaphylaxis has as yet no answer, nor has the question as to whether allergy and immunity are due to the same antibodies.

ALLERGIC FACTORS

Is sudden death an allergic reaction? In 1935 Waldbott[15] published his paper, "The Allergic Theory of So-Called Thymic Death," in which he attempted to prove that in some cases of sudden death the findings in the lungs and other organs were identical with those of anaphylactic shock. Waldbott[16] also suggests that sudden death in an infant with a background of allergy may result from sensitivity to food or other antigens (bacterial or viral) to which extreme sensitivity exists. Hansel[17] discusses Waldbott's work in detail.

Gellis[18] wrote:

> In recent years anaphylactic shock has gained increasing prominence as an explanation for sudden death. Several writers have stressed the fact that infants with eczema are particularly prone to sudden death and feel that this is related in some way to anaphylaxis. However, with increasing care in the isolation of such infants and the use of the newer therapeutic agents, these deaths are apparently less frequent, and it seems fair to conclude that many of these infants succumbed to overwhelming acute infection. The significance of anaphylactic shock cannot be discounted, but its exact role in unexpected sudden deaths remains to be determined.

I feel one of the most important contributions to infant allergies and the allergic mechanism in respect to this problem has been contributed by Clein.[19,20] The work of Clein on cow's milk allergies in infants, and the serious or complicated results therefrom, is original and outstanding. He points out that the type and location of symptoms depend on two factors: (1) the tissue or organ affected and (2) the results of the pathologic mechanism of allergic disease on the particular organ or tissue involved (edema of mucous membranes, spasm of smooth muscle, and excess mucous secretions).

Clein[19] stresses ". . . early recognition and treatment of the allergic state in infants in order to prevent or minimize major allergy later on . . . 82 per cent exhibit their first allergic manifestations within the first four months of life." He lists in order of frequency the following symptoms produced as the result of an allergy to cow's milk or milk products: eczema;

pylorospasm; colic; diarrhea; "very unhappy all the time"; cough, croup, choking, gagging, excess mucus in throat; running nose; constipation; asthma; anorexia; toxemia—apathetic, listless, cyanotic; and urticaria—angioedema. He mentions that the diagnosis is most often missed in infants exhibiting the hazardous group of symptoms—coughing, croup, choking, and gagging, et cetera—the respiratory type.

Clein adds:

. . . The tendency to be allergic is bequeathed these babies by their allergic parents. The degree of intolerance or sensitivity to milk (or other allergens) varies a great deal in each individual; whereas one infant may manifest only a mild colic or "spitting up" when allergic to milk, another may collapse in shock or even die from a few drops of milk placed upon the tongue. . . . It is evident that you cannot divorce the family history of allergy from that of the offspring. It is an important and definite part of the diagnosis.

Since milk is often the only food for the first few months, it is natural to expect some susceptible (allergic) infants to show abnormal manifestations to this food. The toxic reaction produced by milk idiosyncrasy obviously can be serious. Likewise, dangerous or fatal reactions can be produced by other allergens. The important thing is this—the mechanism is the same!

Some infants with allergies have an unusual susceptibility to infectious diseases, and almost without exception such infants are very sensitive to respiratory diseases. Further, it has been my experience, and it is borne out by that of other doctors—both pediatricians and general practitioners—that these children are affected more severely than children without allergies, and they respond to therapeutic measures in a very unsatisfactory manner. Therefore, since respiratory manifestations are so common and so severe in allergic babies, and since the predominant postmortem findings in sudden deaths are found in the respiratory tract, I am inclined to believe that at least some of these sudden deaths can be placed, at least partially, on an allergic basis. I feel that allergy may be *one* key to this problem, and that the respiratory tract may be the shock organ or trigger area.

GAMMA GLOBULIN LEVELS

A new theory has been presented by Spain, Bradess, and Greenblatt[21] that perhaps a lowered antibody level or gamma globulin level or both may be an important factor in sudden and unexpected infant deaths. Since the gamma globulin component includes many of the antibody globulins, this theory is consistent with other evidence as to the natural immunity of the newborn infant, according to Longworth and his coworkers,[22] and it supports my own thinking on the problem.

According to Spain, Bradess, and Greenblatt,[21] the only significant postmortem findings in three infants who died suddenly and unexpectedly were occasional foci of inflammation, particularly in the upper and lower respiratory tract. These areas of inflammation on occasion appeared similar to those seen in the biologic response to infection produced experimentally in the nonimmune animal. Postmortem serum gamma globulin determinations, made 6 hours after death, were found to be below normal for infants of their age period. The gamma globulin levels were 150 mg., 183 mg., and 180 mg., per 100 cc., respectively. These few cases are not sufficient to prove the importance of lowered gamma globulin levels in sudden death, but they do support such a thesis. On this basis they state, ". . . the routine injection of small amounts of gamma globulin in young infants might possibly prevent a substantial number of these sudden and unexpected deaths."

James B. Arey[23] has commented that out of seventy-five infant cases he had studied that came under the classification of sudden death, 68 per cent of which occurred in the first year of life, most were apparently the result of infection. It is his impression that the incidence of sudden deaths runs in almost inverse relationship to that of the normal level of gamma globulin during the first 2 years of life.

Deficiency in gamma globulin, which has been termed agammaglobulinemia or hypoglobulinemia, is thought usually to be congenital. Response to gamma globulin in this condition has not been uniform. Most of the reported cases have been in children in the older age groups who have had histories of multiple infections since infancy or early childhood. Keiden, McCarthy, and Haworth[24] reported a case in an infant 11 weeks old who had generalized vaccinia and staphylococcic septicemia after vaccination against smallpox. The infant was unable to produce antibodies against the virus of cowpox. An absence of gamma globulin was disclosed and in spite of the administration of hyperimmune globulin, the baby died.

Bruton and associates[25] reported three cases of gamma globulin deficiency in patients with histories of multiple infections since infancy. One was treated by monthly injections of gamma globulin, and the patient was maintained free of infections.

Hayles, Stickler, and McKenzie[26] also treated three patients with agammaglobulinemia who had histories of frequent infections since infancy. One of the patients, aged 5 months, died while under antibiotic treatment. The second patient, a 9-year-old boy, continued to have frequent infections and died in spite of the use of antibiotics and the administration of gamma globulin at frequent intervals. The third boy, aged 9 years, was maintained in good health by monthly injection of gamma globulin. These authors state that since their second case did not respond clinically to the gamma globulin in the same manner as the patient of Bruton and others,[25] there must have been some abnormality, as yet unexplained, in the utilization, destruction, or excretion of protein in their patient.

Fried and Henley[27] report a case of a 10-week-old boy in which there was a deficiency of gamma globulin which was accompanied by edema and hypoproteinemia, of possibly *allergic* etiology.

Frawley[28] in 1948 administered gamma globulin to 50 patients with allergic rhinitis (ages not given) during an acute exacerbation of respiratory infection. The results were proportional to the degree of their allergic control.

In those children in whom the offending allergen was known and eliminated, the infectious phase of their respiratory difficulty was improved by a single injection of globulin. In those in whom all the allergens were not known or in whom control was poor, 50 per cent showed considerable improvement for a period of about one month, and in the remainder there was no discernible improvement.

If allergic babies are hypersensitive, susceptible, and immunologically immature, it is a logical supposition that they are hypoproteinemic or have an abnormally low gamma globulin level. Only investigation will prove or disprove this supposition; however, I believe that gamma globulin should be given routinely to *all* newborn babies with a family history of allergy. Better safe than sorry!

A CASE IN POINT

In further support of this thesis, the following case report is cited:

In February, 1954, in working with a pediatrician, I suggested that gamma globulin be given to a new baby to forestall another sudden death in a family where this tragedy had already occurred. On February 20, 1954, the baby was given the first dose of gamma globulin at 1 week of age; the second dose was given March 18, the third April 30, the fourth June 18, and the fifth June 28; all doses were 1 cc. No gamma globulin determinations were made. The usual diphtheria-tetanus-pertussis immunization program was not begun at 3 months since it was felt the gamma globulin would nullify its effects and that the added protection given by the gamma globulin made the chances of any serious illness remote. When the baby was 5 months old, the mother took her away from home for a visit. Several days after arriving at their destination the baby had a dry, hacking cough that suddenly developed into a severe respiratory episode with dyspnea, high temperature, pulse of 180, and a respiratory rate of 80. The baby put up a valiant struggle for 3 days before a diagnosis was made. X-rays were negative. Blood counts convinced the attending physician that the baby had pertussis, although there was no "whoop" at any time. Antipertussis serum (Hypertussis) was given, and within 24 hours the baby was on her way to recovery. There is a strong allergy background in this family.

I think this case history in an infant whose older sibling had died suddenly in infancy constitutes evidence to support the theory that although undue *sensitivity* and *susceptibility* to respiratory infections and insults do run in certain families, the administration of gamma globulin gave this infant added protection and a distinct advantage. She was stricken with a very serious respiratory disease which is often fatal in tiny infants, but she did not go into shock and die; instead she put up resistance and lived.

PREVENTION

How can the problem be summed up? It is obvious that our knowledge at present is fragmentary and speculative and little has been added to clarify the true cause and mechanism of these sudden unexpected deaths of apparently well infants. The most consistent postmortem findings have been in the respiratory tract, diagnosed as interstitial pneumonia or diffuse mononuclear pneumonia, but the causative organism, or mechanisms, have not been definitely determined. The condition is rapid in its course and frequently kills before it is recognized. Nevertheless, progress is being made and the multiple facets seem to be falling into place as more study and research are being done, and more and more men become interested in this problem.

What is the answer? Prevention, or a prophylactic approach, obviously is the solution. Under existing circumstances prenatal immunization of the mother is neither practical nor feasible. Immunization of infants under 3 months is not the answer either, as babies under this age do not respond satisfactorily to antigen stimulation.

The administration of gamma globulin appears to be part of the solution we have been looking for. By using gamma globulin in these infants we give them some of the antibodies and protection needed until they reach the age and stage where the antibody titer and gamma globulin are stabilized at normal levels and until the infant is able to respond to disease processes. The efficacy of gamma globulin as a preventive measure in sudden and unexpected deaths of infants can only be tested by its use in numerous well-controlled cases, both allergic and nonallergic.

The various questions raised in this paper present some of the facets of the problem which have to be resolved. Many investigators are vitally interested in this subject and some are working alone, some thinking alike, and others thinking along different channels. Only through concerted and coordinated efforts, particularly in the large centers with concentrated pediatric services, can progress be made more rapidly in determining the underlying causes of these sudden unexpected deaths in infants. Only when such efforts have been made will we have a more positive approach to the prevention of these deaths.

CONCLUSIONS

1. All newborn babies should have a gamma globulin determination routinely, and all those found to be deficient should be given gamma globulin.

2. All babies with an allergic family background should have the protection of gamma globulin.

3. Sickly newborn infants, regardless of the etiology, should have this added protection until out of danger.

4. A history of a previous "sudden death, suffocation, or unexplained infant death" in a family should be considered an indication for gamma globulin administration to all new babies in this family.

5. Gamma globulin should be given to any child in the above categories as prophylaxis or to modify the potential of any disease process which commonly has respiratory complications—for example, measles—to which they may have been exposed.

6. Further, gamma globulin should be given to any child, regardless of age, who does not respond to antibiotics or chemical therapy while under treatment for an infectious disease process.

7. All babies with chronic gastrointestinal symptoms, or repeated respiratory manifestations, should be suspected as allergy babies and treated accordingly.

8. All obstetrical case histories should include information regarding allergic family traits and/or any unusual prior problems of infancy.

9. Attention should be given and research done in regard to adrenocortical function and its relationship to this problem.

10. My final conclusion is that if every newborn infant, or at least those who qualify as above, were given gamma globulin routinely from birth through the third month, much as we employ other immunization programs, I am convinced that many respiratory fatalities would be prevented and that most of the sudden or so-called suffocation deaths would be eliminated, thus saving the lives of hundreds of babies each year.

Acknowledgment

Sincere thanks are expressed to Edwin B. McLean, M.D., Director of Clinical Investigation, Cutter Laboratories, Berkeley, California, for his encouragement and for his help in collecting the material needed for the preparation of this paper.

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Osteoporosis

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The general framework of the body is built mainly of a series of bones; there are 206 distinct bones in the adult skeleton whose basic primary function is to furnish mechanical support to the human body. Since one of the fundamental laws of biology concerns the interdependence of structure and function, decalcification of the skeleton is increasingly recognized as having an important role. Bone must be considered as living tissue affected not only by the purely metabolic processes of the human organism but also by the mechanical forces of physiologic stress.[1] In order to clarify the nature of decalcification of bone, it is desirable to discuss some of the factors affecting bone physiology.

PHYSIOLOGY OF BONE

It is important to understand that in normal adult life the formation and resorption of bone are in a continual state of flux. Because of the daily wear and tear on the skeleton, definite amounts of calcium and phosphorus are lost and must be replaced. In adult life there is an approximate balance in the two processes; in the child, bone formation predominates, which explains the higher serum alkaline phosphatase levels in children.[2] In the aged, the reverse is true, and bone formation lags behind bone resorption.

Bone formation consists of two processes: (1) the production of the proteinlike organic connective tissue matrix, and (2) the deposition of calcium salts into and upon this matrix.

The bone matrix is a product of the activity of the osteoblasts.[3] These cells are stimulated to form bone in proportion to the degree of activity to which the skeleton is subjected. These cells apparently secrete an enzyme, alkaline phosphatase, which serves as an index for their activity. Siffert[4] has observed that alkaline phosphatase is always abundantly found in the osteoblast and in recently formed bone matrix. Thus the serum alkaline phosphatase levels may be elevated whenever bone matrix is being formed in excess. In order to form this bone matrix, the protein intake and metabolism must be normal. Only in the presence of sufficient and adequate bone matrix can the complex crystals of calcium and phosphorus be deposited to form bone. Dallemagne[5] has demonstrated that most of this is related crystallographically to a complex salt, hydroxylapatite.

There must be then at all times a ready available supply of calcium and phosphorus for daily repair of bone. This naturally depends on their availability in the human diet and on the capacity to absorb them from the intestinal tract. The presence of adequate amounts of vitamin D increases the absorption of calcium from the gut.[6] Since both phosphorus and calcium reach the kidneys via the blood stream, normal renal function is necessary to maintain the normal ratio of calcium to phosphorus. Here the parathyroid hormone takes an active part in controlling the concentration of calcium and phosphorus in the blood stream through its effect on the renal excretion of phosphorus. Parathormone also exerts a direct effect on the osteoclastic cells in bone to increase bone resorption and thus raise the blood calcium level.

Therefore it can be understood that there may be various factors which produce decalcification of the skeleton. Increased function of the parathyroid gland, such as is seen in von Recklinghausen's bone disease, produces excessive bone erosion.[7] Inadequate utilization of vitamin D leads to deficient absorption of calcium from the intestines with resultant failure to calcify the bone matrix, such as is seen in rickets and osteomalacia. It is difficult in this country with its high standards of living to find osteomalacia associated with deficient vitamin D intake. It is probably more frequently associated with chronic steatorrhea, in which there is a loss of vitamins, fat, and calcium from the intestinal tract.

WHAT IS OSTEOPOROSIS?

Simply defined, osteoporosis is bone atrophy. While the rate of bone formation decreases, bone absorption continues unabated with resulting demineralization of the skeleton. Both Albright and Reifenstein[3] and Snapper[8] have emphasized that osteoporosis may result when the production of protein bone matrix is insufficient. Patients with insufficient bone matrix cannot capture the calcium salts to lay down new bone. Since protein bone matrix is a product of osteoblastic activity, a decrease in osteoblastic function may result in demineralization. The normal function of the osteoblasts depends upon the stresses and strains which the body is exposed to as it moves about daily. When the body is immobilized these stimuli stop, and the function

of the osteoblasts decreases. With insufficient matrix formation there is also poor retention of calcium, since there is less tissue in the bone with which the calcium can be combined to form new bone. Consequently, osteoporosis is encountered in clinical medicine in a variety of conditions.

1. Postmenopausal osteoporosis. The skeletal decalcification is localized to the spine, ribs, and pelvis and is associated with deficiency of the steroid-producing glands.

2. In the male climacteric. There may be testicular atrophy or surgical castration with resultant androgenic deficiency.

3. Senile osteoporosis. There is a lack of physical activity. Reduced protein intake may be another factor to be considered.

4. During ACTH and cortisone therapy. Profound effects occur on the protein and mineral metabolism. There is increased nitrogen, calcium, phosphorus, and potassium excretion. Frazier[9] and Demartini[10] have reported pathologic fractures and rarefaction of the skeleton during intensive adrenocorticotrophic therapy.

5. Following prolonged immobilization or disuse. When the body is immobilized, the normal stimulus to osteoblastic activity is curtailed. Snapper[11] has often indicated the danger of renal calculus formation when excessive amounts of calcium and vitamin D are added to the diets of active young patients immobilized in plaster for fractures. Since little bone matrix is being formed during the period of inactivity, little calcium can be deposited in bone. Consequently, large amounts float aimlessly in the blood stream and produce hypercalciuria. The greater the intake of calcium and vitamin D, the greater the amount of calcium absorbed, and the greater the danger that nephrolithiasis will develop during immobilization.

6. Idiopathic osteoporosis. The cause remains obscure; individuals affected are not postmenopausal or senile. Trophic influences affecting the neurovascular nutrition to the skeleton may be responsible.

Albright and Reifenstein[3] consider postmenopausal to be the most common type of osteoporosis. Since the appearance of this form is similar to the other varieties, its clinical signs, symptoms, diagnosis, and treatment will be discussed.

CLINICAL SIGNS AND SYMPTOMS

The general appearance of the patient is usually characteristic; it is that of an elderly female with a spinal dorsal kyphosis lending the appearance of a reduced adult stature. She may relate a history of a gradual onset of weakness, loss of energy, and pains in the middorsal and lumbar spine. She may date the onset of symptoms to an apparent trivial accidental trauma, such as twisting in bed or lifting a stuck window. A sudden fall may herald the onset of radiating pains across the abdomen or down the legs as compression fractures occur readily in the porotic vertebrae, particularly in the lower dorsal and upper lumbar, and in the femora.

Examination of the patient usually reveals a kyphotic dorsal spine with tenderness on palpation over the spinous processes. The entire spine may be rigid with little movement obtained on attempted motion. The skin appears thin and emaciated. The paravertebral musculature is atrophic. Compression fractures frequently are asymptomatic and there may or may not be localized tenderness over them. It is unlikely, in my opinion, that the symptoms arise from osteoporosis per se. They probably follow the onset of complications when vertebral movement becomes fixed and gives rise to referred nerve root pain.

ROENTGEN FINDINGS

Roentgen examination[12] is absolutely necessary for accurate diagnosis of osteoporosis. In early or moderate involvement the shape of the vertebrae may be normal with only sharper than normal definition of the superior and inferior borders. Soon they begin to look "washed out" because they are deficient in calcium. As the involvement progresses, the elastic nucleus pulposus begins to swell and push down on the center of the softened porotic vertebrae, producing an hourglass type of deformity. The intervertebral disks appear higher than normal. The vertebrae diminish in vertical diameter and begin to assume a wedge shape, narrower anteriorly than posteriorly (fish vertebrae). Ultimately compression fractures develop. The major involvement is found in the spine, ribs, and pelvis. The skull shows a normal calcium content except in extreme cases. The extremities also are normal unless the patient has been immobilized for a great length of time, in which case osteoporosis from disuse is superimposed on the skeleton, and, consequently, bone changes occur in the limbs. Interestingly enough, Albright and Reifenstein[3] have pointed out that in postmenopausal osteoporosis the lamina dura of the teeth usually remains intact.

DIFFERENTIAL DIAGNOSIS

Since the roentgen appearance may be similar in osteoporosis and other decalcifying bone diseases, they must be differentiated.

The primary defect in osteoporosis is one of failure of bone matrix formation, rather than of calcium and phosphorus metabolism. Therefore it is not surprising to find a normal serum concentration of calcium, phosphorus, and alkaline phosphatase. Except in acute bone atrophy following prolonged immobilization in young people, higher levels of serum calcium and phosphorus are apt to be found.

Osteoporosis of the spine may be found associated with Cushing's syndrome. The presence of hypertension, insulin-resistant diabetes, moonlike facies, amenorrhea, hirsutism in the female, and high urinary titers of 11-oxysteroids should preclude difficulty in differentiation.

Bone changes seen in primary hyperparathyroidism are usually accompanied by hypercalcemia, hypercalciuria, low serum phosphorus level and elevated serum alkaline phosphatase owing to increased osteoblastic activity in repairing absorbed bone. Early x-ray changes are visualized in the skull and extremities with the typical cystlike areas of resorbed bone. The lamina dura of the teeth disappears early.

Osteoporosis must be differentiated from osteomalacia where the defect is failure of the bone matrix to calcify, rather than failure of matrix formation. Consequently both the serum calcium and phosphorus are markedly lowered.

Another variation of osteomalacia is Milkman's syndrome, in which the bone matrix fails to obtain calcium and phosphorus. It chiefly affects middle-aged women. Bone changes are widespread in the extremities. The disease is characterized by multiple symmetric idiopathic fractures. X-rays of affected areas usually show ribbonlike zones of decalcification or pseudofractures attended by poor healing.

In contrast is the hereditary disease, osteogenesis imperfecta, occurring in childhood and manifested by

multiple spontaneous fractures which heal poorly. The scleras of the eyes appear blue. The serum calcium and phosphorus usually are normal.

Multiple myeloma may produce confusing cystlike decalcifying areas in bone and must be differentiated from osteoporosis. Here again the serum calcium and phosphorus findings may be normal to high so that differentiation must be made by the finding of the Bence-Jones protein in the urine and by the elevated serum globulin in the blood.

In Paget's disease the involved bone may be softened, dense, and cystic so that these various changes going on at the same time produce thickened trabecular patterns interspersed with osteolytic areas. The skull is a frequent site of involvement. Consequently the values for serum calcium and phosphorus are usually normal, but the alkaline phosphatase may be markedly elevated.

Boeck's sarcoid may be differentiated by its tendency to involve the skull and the small bones of the hands and feet, rather than by the generalized decalcification of the skeleton. The blood chemistry shows a high serum phosphatase level with hypercalcemia, hypercalciuria, and hyperglobulinemia. However, the blood phosphorus is usually elevated, and this helps to distinguish the bone changes from those in hyperparathyroidism.

Metastatic bone tumors usually do not offer any difficulty as x-ray reveals individual lesions of the vertebrae which are sharply demarcated from the interspersed areas of normal bone which are invariably found. The serum calcium and the serum phosphatase levels may be elevated if the bone lesions are extensive. A primary source is to be looked for and is most apt to be found in the breasts, kidneys, prostate, or lungs.

TREATMENT

In the treatment of postmenopausal and senile osteoporosis, major emphasis has been placed on the use of the gonadal hormones. Albright and Reifenstein[3] have studied the effect of androgens and estrogens on patients with postmenopausal and senile osteoporosis and have demonstrated the following:

1. Androgens (testosterone propionate and methyl testosterone) produce a marked decrease in the excretion of nitrogen, calcium, and phosphorus in the urine and the stool.

2. Estrogens (natural and synthetic) also produce a marked decrease in calcium and phosphorus excretion, but with less marked effect on the retention of nitrogen.

3. The effect on calcium metabolism of estrogen and androgen in combination is greater than the effect of either alone in postmenopausal and senile osteoporosis.

Shorr and Carter,[13] under carefully controlled metabolic experiments, have demonstrated the value of the administration of strontium lactate and vitamin D_2 as an adjuvant to calcium in the remineralization of the skeleton. They showed that when the daily ceiling for calcium storage is reached (1.5 to 2 grams), the addition of strontium results in greater retention. Maximal retention of strontium and calcium is achieved with the use of auxiliary agents, that is, vitamin D, estrogens, and androgens.[14]

Routine treatment consists of:

Androgens: (1) Methyl testosterone 40 to 100 mg. daily or (2) testosterone propionate 25 to 50 mg. three times weekly intramuscularly. Although the same metabolic effects on nitrogen retention were observed by Albright and Reifenstein[3] with the oral route of administration, Snapper[11] favors the intramuscular route.

Estrogens: (1) Premarin, 1 tablet (25 mg.) three times a day by mouth, or diethylstilbestrol, 0.5 to 1 mg. daily, or ethinyl estradiol, 0.05 to 0.02 mg. three times a day, or (2) estradiol benzoate, 10,000 R.U. (1.66 mg.) intramuscularly three times weekly.

Diet: (1) A diet rich in protein plus a daily intake of 1.5 grams of natural calcium (about 3 pints of milk provide this ration). (2) Shorr and Carter[13] recommend a total daily intake of 6.4 grams of strontium lactate in three divided doses, as the combined retention of calcium and strontium is far greater than the retention of calcium alone, no matter how high the calcium intake. These amounts have been given over a period of 4 years without evidence of toxicity. The addition of 50,000 units of vitamin D_2 daily will increase the absorption of calcium and strontium from the intestines.

CONTROL OF SIDE EFFECTS

Unfortunately steroid therapy may cause sodium retention in elderly patients with resultant edema. A low sodium diet and/or ammonium chloride may be indicated to overcome this effect. Signs of masculinization in the female such as husky voice, excessive hair growth on the face, and acne, may follow prolonged high dosage of androgens. The simultaneous administration of estrogens has a tendency to minimize the signs of virilism. Estrogens should be given for 21 days with a rest period of 1 week in order to avoid any possible carcinogenic effect. Occasionally, withdrawal bleeding is encountered which ceases with continued administration of androgens. In old men, testosterone should be used cautiously because of its stimulatory effect on the prostate; the prostate should be checked at frequent intervals for evidence of enlargment or change in consistency. Estrogens have a tendency to minimize these adverse effects.

EFFECTS OF MOBILIZATION THERAPY

Whedon, Dietrick, and Shorr[15] have conducted some interesting metabolic experiments on normal adult men, immobilized for a long time, by placing them in the Sanders oscillating bed. The bed allowed regular rhythmic movements of these patients and resulted in reduced loss of nitrogen, calcium, and phosphorus. This suggests the desirability of a procedure which will maintain the body in the best possible metabolic and physiologic state during any period of inactivity or immobilization. There appears to be a positive correlation between the degree of inactivity and osteoporosis.

As individuals pass middle age, they tend to become less active, either by choice or from the crippling effects of chronic illness. These people should have the benefits of regular osteopathic manipulative therapy with mobilization of all spinal facets. They should engage in free active use of their extremities whether confined to bed or chair. Even in cases of compression fractures of osteoporotic vertebrae, it is not advisable to confine the patient to bed for prolonged periods. A light spinal corset or brace should be fitted for support and the patient mobilized.

CRITERIA FOR RECOVERY

Most cases of postmenopausal osteoporosis respond favorably to therapy; back pain disappears and there is restoration of the sense of well being. However, it is difficult to demonstrate the recalcification of osteoporotic vertebrae on the roentgenograms. Most clinicians express doubt that sufficient changes are

shown in the x-rays to serve as criteria for cessation of therapy. Snapper[16] continues androgen therapy after subjective improvement has been obtained. He usually prescribes one or two injections per week for an indefinite time. Even if the patient is asymptomatic and the osteoporosis discovered on routine spine and pelvic studies, with no involvement of the extremities or skull, he is in favor of starting treatment with androgens.

SUMMARY

Bone formation consists of the manufacture of a proteinlike matrix in which a deposition of calcium salts occurs. This metabolic process is dependent on various factors: availability of essential minerals and vitamins, adequate protein intake and normal synthesis, and the stimulus of osteoblastic activity by the daily movements of the body.

Osteoporosis results when the rate of bone formation decreases and bone resorption continues unabated. It develops (1) in the postmenopausal state, (2) in the male climacteric, (3) in senility, (4) during ACTH and cortisone therapy, (5) following prolonged immobilization, and (6) from obscure causes, that is, idiopathic osteoporosis.

The clinical symptoms of postmenopausal or senile osteoporosis are spinal pain or discomfort in the middorsal and lumbar region with the development of radiating pains particularly after compression fractures of the vertebrae.

Roentgen examination is essential for diagnosis and usually reveals a "washed-out appearance" of the spinal vertebrae. They assume an hourglass shape with higher than normal intervertebral disk spaces. Compression fractures of the lower thoracic or upper lumbars may be present with anterior wedging of the bodies.

Postmenopausal osteoporosis must be differentiated from primary hyperparathyroidism, osteomalacia, Milkman's syndrome, osteogenesis imperfecta, Paget's disease, Broeck's sarcoid, multiple myeloma, and metastatic tumors.

Treatment consists of combined androgen and estrogen therapy with concomitant administration of strontium lactate and vitamin D_2. Mobilization of the patient and osteopathic manipulative therapy are definitely indicated.

120 Remsen St.

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Operative Obstetrics*

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Although the field of operative obstetrics is vast, this paper will deal primarily with three phases, namely, forceps extraction, internal podalic version, and cesarean section.

The history of operative obstetrics shows many wide swings of the pendulum from conservative treatment to liberalism. Modern treatment would have been frowned upon only a few years back.

In general, all types of operative obstetrics are directed toward maternal and/or fetal salvage. The clinical situation at hand must be evaluated from the standpoint of both. The obstetrician must weigh the various procedures and select the one that gives the greatest prospect of a successful outcome.

FORCEPS EXTRACTION

The obstetric forceps had been originated by the sixteenth century, but it was not in general use until the eighteenth century. Since that time, many variations have been made in these instruments, but they all have the same basic principles.

It is well known that the forceps has four principal uses. It is used as (1) an extractor, (2) a rotater, (3) a compresser, and (4) a lever. Of these, extraction and rotation are most important, with lever action next. The use of compression is employed only to a minor degree in good obstetrics.

Prequisites to Operation.—

Prior to any type of forceps application certain conditions should be determined:

1. Cephalopelvic disproportion must not exist.
2. The cervix should be fully dilated or easily dilatable.
3. The membranes should be ruptured.
4. The position of the head must be known exactly.
5. The bladder and bowel should be empty.
6. The baby should be alive and have a reasonable chance of survival.

Failure to observe these conditions can only lead to harm which may be disastrous to both mother and child.

Indications.—

The indications for forceps extractions are numerous. They might be grouped by some individuals

*Presented at the Fifty-Eighth Annual Convention of the American Osteopathic Association, Toronto, Canada, July 14, 1954.

as those of necessity and those of convenience. Forceps of convenience has no place in osteopathic obstetrics and is used only by those who have no respect for the mother or the future welfare of the infant.

The indications for forceps of necessity will vary with different clinicians, as rarely will two obstetricians agree on every case involved. Therefore, only the most commonly accepted indications will be mentioned. They may be classified as maternal or fetal, and some will be both.

Maternal indications are those of intercurrent diseases such as toxemia, infection during labor, placenta previa, abruptio placenta, tuberculosis, cardiac and renal conditions, and such diseases as would interfere with or jeopardize the mother after the second stage of labor. It should be remembered that the expulsive effort of the second stage may place considerable demand on the mother's resources, and, in the above conditions, it may be desirable to eliminate this drain. Uterine inertia and other failures of the expulsive powers constitute approximately 75 per cent of the indications for forceps delivery.[1] Objective maternal exhaustion and arrest of the progress of labor in the second stage can be readily diagnosed. Slow progress of labor should not be considered an indication for interference. Maternal exhaustion, of course, can best be determined from the increased pulse and the temperature and blood pressure changes.

Fetal indications include prolapsed cord, fetal exhaustion, and failure of proper rotation of the head. With prolapse of the cord, immediate delivery is advisable if the child is alive, the cervix dilated enough for forceps extraction, and the head advanced to or past the zero station. In a high station, conservative treatment or internal podalic version could be the procedure of choice. When signs of fetal exhaustion appear, such as a persistent increase or decrease of the fetal heart rate or the passage of meconium, forceps extraction should be done. In slow progressive labors, where it is felt that excess intracranial pressure is being developed through the prolonged compression and congestion, immediate delivery is indicated.

Arrest of rotation of the head, such as occipitoposterior or transverse, often demands the use of forceps. A slowly rotating head should not be considered an arrest. If the head is engaged and no descent or change in position is made with 2 to 4 hours of active labor, the head may then be considered in a state of arrest.

Diagnosis of Position.—

The position of the fetal head should be known before any forceps application is made. The position can be determined from the sagittal suture, the fontanels, and the ears during a sterile vaginal examination. In some instances, there may be a large caput succedaneum and even overlapping of the skull bones which may be confusing. In these cases it will be necessary to remember the number of sutures that radiate from each fontanel—three from the posterior and four from the anterior.

Palpation of an ear may clarify an otherwise obscure diagnosis. A diagnosis based upon the position of the baby's ear is nearly foolproof. After the ear, which points to the occiput, or the tragus, which points to the face, has been located, the necessary direction of rotation will be known.

In the lower stations of transverse positions, the anterior ear can usually be felt with one finger. The index finger, with palm up, is inserted between the fetal head and the symphysis, and the ear palpated with the fingertip. If the head is in the midplane or higher in the pelvis, the posterior ear can be felt by passing the hand or fingers, with palm upward, along the perineum toward the sacrum.

The higher the forceps application is to be made, the more important it is to know the exact position of the head. A mistaken diagnosis can result in serious damage to the baby.

Choice of Forceps.—

Of the various forceps in use today, I prefer to use either Kielland or Luikart forceps for the majority of cases.

With the introduction of Kielland forceps, a principle and not primarily an instrument was developed. This forceps differs primarily from other models in that the bend is at the junction of the blades and the shanks, thus eliminating the conventional pelvic curve. This in effect straightens the long axis and increases its value in rotation.

The Luikart forceps consists of the standard pelvic curve with unfenestrated blades. The use of this solid blade has greatly reduced the possibility of injury to the fetal structures, especially about the face.

Both of these forceps employ the sliding handle which gives the operator an opportunity to bring the fetal head into synclitism. Usually with the first traction the head becomes synclitic. Many physicians fail to recognize this important maneuver and that it is much easier to extract the fetal head with all of the smallest diameters presenting themselves.

Technic.—

Forceps application may be classified as high, midplane, low, and outlet. The procedure for outlet forceps has not changed much in the past decade, and further discussion does not seem necessary.

Failure of rotation of the fetal head or other cephalic malpositions can present at times a most difficult situation in obstetrics. The most common of these are occiput transverse and occiput posterior positions. Fortunately, most occipitoposterior positions will rotate anteriorly if given ample time.

The management of posterior positions as described by Whitacre[1] is a practical approach to this problem. Failure of rotation of the head with arrest of labor demands rotation of the head before extraction, or internal podalic version with extraction.

Manual rotation should be attempted first when such a condition exists. One hand, usually the left, is inserted in the vagina for right-sided presentations. For left positions the right hand is used. The head is grasped with the thumb on the anterior malar bone and the fingers on the posterior parietal area. The head is raised slightly in the pelvis and rotated a few degrees in the direction desired. At the same time, the opposite hand grasps the fundus of the fetus in the area of the anterior shoulder, and rotation of the body and fetal head is accomplished. Caution is necessary in raising the head, for elevating it to the pelvic inlet would present further problems.

Even if complete manual rotation fails, the head can at least be advanced to a transverse position. In this position rotation and extraction can be completed with forceps.

In a transverse presentation Luikart forceps can be applied in the anterioposterior diameter of the pelvis. In right-sided positions the left blade is inserted first. This blade is inserted with the handle pointing directly above the symphysis and with the blade next to the

posterior parietal area. It is much better to insert only about 1 inch of the blade than to insert the entire blade. The blade is then rotated over the brow to an anterior position where the handle is depressed. From this point the blade may be pushed up over the side of the head. The right blade is then inserted in a posterior position. Locking should easily occur unless some soft parts have been caught.

With this method injuries to the face are avoided. If full insertion of the left blade were accomplished prior to placement over the brow, the blade would of necessity move over the face. This might cause the head to shift back to its posterior position because the forceps blade meets with the nose and other irregularities of the face.

After locking the forceps the head is rotated a few degrees at a time, with gentle traction being applied, until it reaches the anterior position. At this time a slight readjustment of the blades may be necessary in order to secure an ideal biparietal application. The head is then extracted with downward and forward traction, bringing it through the canal with the same motions as in a normal delivery.

In left occipitoposterior positions the same technic, with modifications, can be used. The left blade is inserted first, in this case posteriorly, and the right blade is rotated over the brow and inserted to its normal position.

Direct occipitoposterior positions can be managed in a similar manner, the head being manually rotated to the transverse position, the forceps applied, and the head rotated as previously described, with complete extraction. Consideration can also be given to delivering the head as an occiput posterior if the rotation is not easily accomplished.

The use of Kielland forceps as discussed by Titus[2] offers some advantages in the management of transverse and posterior occiput presentations. With the Kielland forceps a different technic is involved in the management of these positions.

The anterior blade is inserted first, and in a right occiput transverse presentation the left blade is anterior and is applied first. It is inserted anterior to the head in a spoonlike manner with the concavity of the cephalic curve upward. This is passed between the head and anterior lip of the cervix and pushed upward into the cavity of the uterus until the junction of the blade and shank is above the symphysis. The left blade is turned gently toward the mother's left (clockwise) until the concavity of the cephalic curve is down. The blade is then pulled down over the head.

The direction of rotation of the anterior blade is most important, as turning in the wrong direction may injure the lower uterine segment. To aid in the prevention of this mistake, the manufacturers have placed either a button or an arrow on each handle, and rotation is always in the direction of the button or arrow. It should be remembered that in the application of these forceps the right blade is always rotated to the mother's right and the left blade to her left.

The right blade is then inserted posteriorly in a normal manner. After application, the forceps is locked and adjustment made with the sliding lock when the forceps is adapted to the head. The head is rotated in a clockwise manner to the anterior position where it is extracted by following the normal birth channel, the suboccipital region being brought under the symphysis, and the head then extended.

Episiotomy should be done in forceps extraction procedures as well as with internal podalic version and extraction. I prefer the midline incision in the management of these cases.

INTERNAL PODALIC VERSION

Although internal podalic version and extraction constitute perhaps the oldest known obstetric operation in use today, this operation offers hazards for both mother and child. The resultant mortality of this procedure restricts its general use. Erving and Kenwick[3] have reported a fetal mortality as low as 5.5 per cent. The dangers are minimized in the hands of experts, but with the occasional operator fetal deaths may run as high as 50 per cent.

Because of the high fetal mortality, it is obvious that this operation is not one of election. Podalic version should be reserved for cases of transverse presentation and possibly prolapse of the cord.

Internal podalic version is usually combined with extraction of the fetus; however, when only partial extraction is done it is known as a Braxton Hicks version. The pulling down of one or more extremities has been used in the management of placenta previa. This usually results in the loss of the baby, and the obstetrician should consider carefully before he decides to perform this maneuver.

Prerequisites.—

The prerequisites for this procedure are the same as those for forceps application except that usually a deeper general anesthetic is necessary. The membranes should be unruptured or only recently ruptured. This affords a greater laxity of the uterus and allows more room for movement of the baby. If the membranes have been ruptured too long, the uterus contracts about the fetus and does not leave ample room for the turning procedure. The head must be readily displaceable from the pelvic inlet.

No cephalopelvic disproportion should exist. The uterus must not be spastic or a high retraction ring present. The operation is contraindicated in hydrocephalus and should not be attempted under too light an anesthesia.

Indications.—The indications for internal podalic version are usually malpresentations such as transverse or oblique positions, poorly engaging face and brow presentations, multiple births, prolapsed cord, and, occasionally, occipitoposterior positions.

Technic.—

The technic of internal podalic version from the vertex is that described by McElroy.[4]

With the patient under deep surgical anesthesia the operator's hand, palm up, covered with an elbow-length glove, is inserted through the dilated cervix into the uterus. The head is grasped and pushed up out of the inlet and to one side.

The hand is then passed upward into the uterine cavity until the feet are reached. They can be distinguished from the hands because the toes are in a straight line while the fingers are in a pointed line. Palpation of the heel and the palm will also aid in differentiation. If on palpation the operator does not have intrauterine clearance for his fist, version may be difficult or impossible.

Either one or both feet are grasped and pulled downward. The head is kept elevated as traction is placed on the lower extremities. In the performance of this procedure the long axis of the fetal ovoid must cross the long axis of the uterus at right angles. This

will form a resistance point until the rotation has passed the halfway mark. Gentle pressure should always be used, as excessive pressure or haste may rupture the uterus.

The baby, via the legs, is rotated so that the back is anterior. After delivery of the legs, the operation is completed as a breech extraction, the arms being pulled down, head flexed, and delivered. Aftercoming forceps may be used on the head.

In order to prevent prolapse of the cord, the intrauterine hand should not be removed once the operation has begun. After the placenta has been delivered the uterine cavity should be explored for lacerations or rupture of the uterus.

CESAREAN SECTION

Today's concept of the employment of cesarean section is rapidly changing from that of even 10 years ago. Cesarean section about two decades ago was used more liberally as a method of terminating labor, and along with this came a rise in maternal and fetal mortality. This rise in maternal mortality produced a change in obstetric thinking, and the tide turned in the other direction.

Within the past decade, broadening of indications for cesarean section has been made possible by the successful employment of antibiotics and improved facilities for prompt replacement of blood. In addition, adoption of improved surgical technics with superior anesthesia methods and the employment of more rational supportive therapy have contributed to increased safety of the operation. A better understanding of the fluid electrolyte balance and administration of fluid electrolytes at the proper time have helped to prevent the development of derangements.

Prerequisites.—

Several recognized rules with regard to cesarean section must always be on the obstetrician's mind. First, it should be remembered that cesarean section is not to be considered as strictly an emergency procedure. Poor obstetric judgment has been used when a woman is faced with the necessity of abdominal delivery toward the end of labor. Second, and possibly more important, it should be remembered that after a preliminary 6- to 8-hour latent period, the mortality rate of section rises with every hour the woman is in labor. Third, and not least, is the general rule that in the presence of actual infection, section is best followed by hysterectomy. Many operators have been "lulled to sleep" by the fabulous claims for antibiotic agents.

Indications.—

The indications for cesarean section are numerous. In general, one might class the indications in two categories, maternal and fetal.

The Committee of National Osteopathic Obstetrical Statistics of the American College of Osteopathic Obstetricians and Gynecologists[5] reported a series of 114,902 deliveries from some of the osteopathic hospitals between 1950 and 1952. Of this number 7,005 were cesarean sections. The five most common indications for section were:

Cephalopelvic disproportion	30.53 per cent
Previous section	21.54 per cent
Placenta previa	6.77 per cent
Toxemias of pregnancy	5.15 per cent
Abruptio placentae	2.7 per cent

Some of the other common indications that appeared in this report were prolapsed cord, elderly primigravids, uterine inertia, transverse presentation, cervical stenosis, ruptured uterus, tumors of the uterus, diabetes mellitus, cardiovascular diseases, and prolonged labor.

Technic.—

As in all surgical procedures there are numerous types of sections, the oldest of which is the classical section to which various modifications have been applied.

In my opinion, the low cervical section is the preferred procedure. Whether a transverse or longitudinal incision is made in the low cervical segment does not actually matter, as both procedures have their advantages. This procedure can be utilized after a test of labor and even after careful vaginal examinations have been made, whereas the classical procedure would be dangerous.

The cervical route affords greater protection against peritoneal contamination or infection. The bladder flap closing over the wound affords better peritonization, and the cervical structure gives a firmer closure. By this method, any spillage of amniotic fluid is limited to the lower portion of the abdomen. Hemorrhage is usually less, as the venous engorgement of the cervical segment is not so great as that of the fundus. The muscle fibers of the lower segment tend to heal faster and give a stronger union with less possibility of rupture in a future pregnancy.

Shock is not so apt to occur since the omentum and intestines are not interfered with as much as in the classical procedure.

Postpartum complications, such as ileus, peritonitis and gastric dilatation, are rarer. The closure is better because it is protected by the bladder flap which limits the possibility of seepage of the lochia. With early ambulation, the lower segment is more at rest and does not tend to flop and permit oozing of exudate throughout the abdomen.

CONCLUSION

This discussion has been offered to explain a few of the current operative obstetric procedures. Proper application and use of forceps will not injure the baby or the mother. Internal podalic version and extraction have a definite place in the hands of the well-trained obstetrician and involve a minimum risk in selected cases. This procedure should be shunned by the occasional operator. In the present era of chemotherapy and antibiotic therapy, abundant blood transfusions, and relatively standardized safe technics, cesarean section also offers a relatively safe means of terminating pregnancy.

Modern obstetrics calls for the physician to choose a procedure that will not injure the mother or the baby.

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Diagnosis and Treatment of Bronchial Asthma*

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"All is not asthma that wheezes"[1] points out the importance of an adequate history as the first step in the diagnosis of bronchial asthma. It is not uncommon to find the patient symptom free after a paroxysmal or primary attack of bronchial asthma. This paper will consider the diagnosis and treatment of this entity in children.

The fundamental principles of allergy in children are somewhat different from those of allergy in adults. Heredity or constitutional predisposition is a much greater factor. It is not uncommon to find that one or both parents of an allergic infant or young child have a positive history of allergy. The fact that eczema or asthma was present at birth, or occurred shortly thereafter in breast-fed infants, supports the theory of heredity. Discussion of whether this is due to placental transmission, is germinal in character, or is due to overindulgence in certain foods by the pregnant or lactating woman is not the purpose of this paper. Of clinical value is the knowledge that the young child is more likely to be sensitive to foods than to other allergens,[2] whereas inhalants (particularly dust), drugs, and bacteria are of relatively and increasingly greater importance in older children.

HISTORY

A detailed history of the circumstances concerning the onset of the bronchial manifestation is important because it often suggests the possible causative factors.[3] The nature of the individual attacks and their frequency and duration must be evaluated. Are the attacks intermittent or continuous; do they have a definite relation to the time of the day or night; and under what circumstances do they occur?

In pediatrics bronchial manifestations are not infrequently preceded by a gastrointestinal allergy, eczema, or urticaria which appeared in infancy or early childhood. Bronchial asthma may be precipitated by some of the acute infectious diseases such as pertussis, measles, diphtheria, scarlet fever, and the common cold, especially in those children who have some manifestation of allergy. The bronchial symptom may be preceded by sinusitis and develop into an allergic state.

A change in diet or the addition of a new food may suggest a possible offender. A change of residence or climate or the introduction of new contacts into the surroundings, such as furniture, rugs, clothing, or pets, may be the source of potent allergens. Proximity to stables with their horses, cattle, or chickens may be the exciting factor.

Although individual articles of furniture or other accessories may be the chief or individual sources of allergens responsible for the patient's symptoms, house dust, a mixture of a great variety of allergenic materials, is a most important factor to be considered in those patients sensitive to animal emanations, cotton linters, and cottonseed.[3] This is especially true of children who spend a great deal of time in the house.

The time of the onset of symptoms may give a clue to the cause. If they manifest themselves during the night, the allergens may be present in the bedding, rugs, carpets, furniture, draperies, or in the stuffed toys the child takes to bed.

Contributing Factors.—

Contributing factors are important not only in diagnosis but also in the clinical management of the problem.

Psychosomatic factors, which must be considered as contributing factors, may be explained as a stress reaction.[4] However, the psychosomatic state of the patient may be caused by the repeated attacks of bronchial asthma.

Swineford and Weaver[5] believe that a great many physical stimuli play an important part in the clinical course of allergy; they are "triggers" rather than primary causes. The following may be of significance, particularly in respiratory allergy: draughts, wet feet, temperature changes, humidity, dampness, going out after bath, swimming, water, heat, cold, chilling, sun, exercise, fatigue, excitement, bathing, cooling after exercise, loss of sleep, pressure.[3]

Osteopathic structural lesions in the upper dorsal area and ribs may be a primary or secondary factor of great importance. It cannot be overemphasized that the child treated with osteopathic manipulative therapy has less respiratory difficulty and is less apprehensive.

Disturbances of the endocrine glands may be contributing factors. Hypothyroidism or hyperthyroidism may play a role. Pituitary insufficiency is not infrequent in allergic children.[3]

The nutritional state of the patient may be of definite significance in the clinical course of the disease. Loss of weight is common in severe bronchial asthma. A restricted diet prescribed as the result of a suspected food allergy, or loss of sleep may be factors. The eating habits of the patient must be questioned, because many patients eat improper food, and the diet is poorly balanced. If there is any one chief parental complaint in pediatrics, it is the dietary habits of the child. Relief of the bronchial attacks, better sleep and rest, and a properly balanced diet with sufficient calories and vitamins produce a gratifying change in the nutritional state of the patient.

CLINICAL MANIFESTATIONS OF BRONCHIAL ASTHMA

The physical signs found on examination will depend largely upon the time at which the examination is made and upon the duration and character of the asthmatic attack.[2]

Fortunately, bronchial asthma is comparatively rare during the first 2 years of life.[6] According to Buffum,[7] the diagnosis of asthma in infancy should, if possible, have three foundations as its basis:

1. Recurrent wheezing should be present. By wheezing is usually meant bronchial dyspnea with prolonged expiration and musical rales. Unfortunately, infants do not always have the typical asthmatic rales; instead they have noisy difficult breathing, and the musical expiration is drowned out by the mucus in the upper respiratory tract which produces a loud gurgling sound.

2. Allergy should be demonstrable. Substantiation may be had from a family history of allergy, history of eczema, allergic rhinitis with pronounced eosinophilic

*Presented at the National Osteopathic Child's Health Conference and Clinic, Kansas City, Mo., April 4, 1955.

discharge, positive results of skin testing, asthma, or other manifestation produced by some inhalant or food. A history suggestive of allergic factors and a recent blood count in which the differential shows an eosinophil content greater than 4 per cent is of value in confirming the diagnosis of bronchial asthma.

3. There should be no other condition or disease which could cause the wheezing, such as pneumonitis, tuberculosis, foreign body, heart disease, especially congenital abnormalities of the blood vessels or trachea, and cystic fibrosis of the pancreas.

In many infants the required evidence is lacking; the wheezing is not characteristic, and the allergic state is difficult to demonstrate. A good percentage do not have eczema, and there are no eosinophils in the nasal or bronchial secretion. In others results of the skin tests are negative or insignificant. In still others considerable time is required to identify food factors and even inhalants. In such infants the physician must be satisfied to make a tentative diagnosis and then work out a more definite diagnosis over a period of weeks or even months. Confirmation is aided when, by elimination of the suspected food, marked improvement results and, not uncommonly, symptoms return with reintroduction of the eliminated food. Wheezing which disappears on admission of the infant to the hospital and, without any change in management, recurs when the child returns home also helps make the diagnosis definite. Skin tests are not usually positive until after the second year.

During childhood, the outstanding symptom of bronchial asthma is shortness of breath or dyspnea accompanied by a musical rale or whistling which is known as the wheezing type of respiration. It may have a sudden or insidious onset. Very often the attack occurs suddenly without any warning symptom. When this is the case the patient complains of a smothering feeling in the chest and has great difficulty in breathing. He attempts to assume the most comfortable position, one which provides the greatest amount of air exchange with the least effort. He is usually found leaning forward while sitting up in bed or on a chair. During this phase the respirations are slow and labored, sweating is marked, the facies is anxious, and the patient appears in great distress. Respirations are strictly thoracic, and the thorax appears distended. The lip and nail beds appear slightly cyanotic. The patient is usually afebrile.

Next to the dyspnea and expiratory wheezing, cough is the most important symptom and is practically never absent. It may precede the attack, but if the onset is sudden, it usually follows. At the onset the cough is short and dry or deep and harassing. At times it may be continuous and further exhausts the patient; the pulse is then weak and rapid, and the patient appears to be unable to speak.

As the attack lessens, the patient may expel a mucoid plug which may terminate the attack. The cough later becomes looser and less irritating, and expectoration is freer. With the disappearance of the initial symptoms of dyspnea and expiratory wheezing, the cough may disappear, but it may last for a few days or for weeks.

When the attack is ushered in less suddenly, there are usually premonitory or warning symptoms such as nasal congestion with watery discharge accompanied by sneezing, which may last for several hours or days. The nasal mucous membranes are pale pink, and there is usually a slight cough. At this time the child is thought to have a cold or mild upper respiratory infection. He is usually afebrile, but in the presence of infection, he may have a fever. Musical rales may be present at this time, and the patient not uncommonly complains of heaviness in the chest with some difficulty in breathing. As the bronchial asthma becomes more apparent, the symptoms are those of the attack with a sudden onset. On percussion the chest is hyperresonant. On auscultation the breath sounds are weak, usually as the result of the little exchange in air, and not too much wheezing will be heard. The sibilant and musical rales often drown out the breath sounds. Occasionally atelectasis results from plugging of a bronchus with tenacious mucus, and the mediastinum is then found to be shifted toward the affected side. In prolonged attacks the heart may fail as a result of existing emphysema; fortunately this is uncommon in pediatrics. As the attack subsides the symptoms are not as marked; the breath sounds become more audible, and the musical rales are not as noticeable. Respiration is deeper, breathing tends to be easier, and the abdomen is not so tense. Cough becomes productive. If treatment is effective, the attack may terminate abruptly, or it may continue with less intensity and with exacerbations. Recurrence of attacks is characteristic of bronchial asthma.

LABORATORY AIDS TO DIAGNOSIS

Fluoroscopic examination usually reveals the diaphragm to be low; its excursions are decreased, especially during an attack.

A complete blood count usually reveals eosinophilia, and there is a marked increase in eosinophils in the sputum and nasal smears.

Skin tests are important, but their technics will not be discussed in this paper. The patient must be tested for all possible inhalants and for the foods eaten.[7] Important allergens may be missed if tests are incomplete. Scratch tests should be performed first to prevent possible systemic reactions. Intradermal tests should be resorted to if insufficient information is obtained by the scratch method. It is not advisable to use group testing as reactions may be missed as a result of dilutions. Testing, of course, must be correlated with the history of exposure and with clinical evidence.

DIFFERENTIAL DIAGNOSIS

Bronchial asthma, particularly the mild form or premonitory symptoms, may have to be differentiated from the common cold. The clinical manifestations of the common cold in the infant and younger child differ somewhat from those in the older patient. During infancy, fever is an outstanding symptom and is usually moderate or high, while in the older child it is low grade. The infant usually shows marked irritability and restlessness, vomiting or diarrhea is not uncommon, and there is frequently vascular congestion of the ear drum. The other manifestations are the same regardless of age: nasal and pharyngeal irritation, sneezing, watery nasal discharge becoming thicker in a few days and finally purulent, and reddening of the nostril and upper lip. The nasal mucous membranes are angry red, and there is an absence of eosinophils in the discharge and if a complete blood count were performed there would be 2 to 3 per cent eosinophils. In repeated colds the proof of the diagnosis rests in the absence of a specific allergen.

Recurrent attacks of acute bronchitis in association with acute respiratory infection may be extremely difficult to differentiate from bronchial asthma. These

patients have a certain amount of respiratory wheezing and dyspnea as well as musical rales on auscultation. They do not have any allergic history or basic allergic factors. There is an absence of eosinophilia, and relief is not obtained with the administration of epinephrine. During the first attack it is practically impossible to make a differentiation; fortunately, as the child grows older the attacks disappear. The rales are usually coarse, moist, and prominent over the lower posterior portion later in the disease.

Allergic bronchitis may have to be differentiated from bronchial asthma. The child with allergic bronchitis has recurrent paroxysms of unexplainable coughing without the asthmatic wheeze which last for a considerable time. This is probably better termed an "allergic cough," and if treated as such, it can be relieved.

Aspirated foreign bodies may cause symptoms which are confused with bronchial asthma. When aspiration takes place in the presence of an adult, and when the symptoms of violent coughing and dyspnea are immediate and severe, the diagnosis can be easily made. But when no one sees the foreign body aspirated and the immediate symptoms are not alarming, the diagnosis becomes more difficult.

The symptoms depend on the size of the foreign body and the site in which it lodges.[8] Recurring fits of coughing may be the cause for the visit to the doctor, and in the absence of other definite signs in the lungs, the condition may be diagnosed as whooping cough or pressure from enlarged glands, as of the hilar lymph glands in tuberculosis. The foreign body may have already produced local bronchopneumonic changes in the lungs and the primary cause may be overlooked. In such a case differentiation from asthma may be made only with the assistance of x-ray studies of the lungs. If x-ray studies reveal a radio-opaque foreign body, and whooping cough or other pneumonopathy is not present, bronchoscopic examination should be made. If the foreign body is not coughed up or removed, cough may occur in paroxysms. The foreign body may shift its position and result in attacks of dyspnea, cough, cyanosis, and hemoptysis,[8] or it may imbed itself in the mucous membranes and not give rise to symptoms until infection sets in, when swelling of the mucosa and secretions lead to obstruction, collapse, and bronchiectasis in the lung beyond, or possibly to a lung abscess.

Enlargement of the thymus gland must also be considered in the differential diagnosis of bronchial asthma. Thymic asthma, in young infants, particularly in early infancy, as a result of compression of the soft trachea by the enlarged thymus, produces stridor and sometimes cyanosis. Diagnosis is made by x-ray by a competent radiologist and should not be attempted without it. The associated asthma-like symptoms with the x-ray findings make the diagnosis. X-ray therapy results in diminution of the thymus and disappearance of symptoms without recurrences.

Tuberculous and nontuberculous tracheobronchial lymph nodes must be differentiated from bronchial asthma. When the glands are sufficiently large to cause symptoms, it is usually before the second year, and the dyspnea as the result of bronchial compression is not relieved by subcutaneous injection of epinephrine. The positive chest x-ray findings, and positive tuberculin reaction in the case of tuberculous lymph nodes, confirm the diagnosis.

Bronchial asthma may have to be differentiated from cystic fibrosis of the pancreas (mucoviscidosis). Symptoms are present in very early infancy in this condition which has an insidious onset. The first bronchial symptom is frequent and persistent cough. The first evidence of digestive difficulty is the failure to gain weight; moderate diarrhea may be present at the onset but usually the stools are foul and bulky. The mucus-secreting glands of the entire body are affected in addition to those of the pancreas which account for the gastrointestinal and nutritional symptoms.[9] The tenacious mucus in the bronchi leads to obstruction emphysema or atelectasis and bronchopneumonia. The infant shows a marked decrease or absence of pancreatic enzymes in the stools or in secretions obtained by duodenal aspiration. X-ray findings of the lungs are characteristic of the disease.

Acute laryngotracheo bronchitis may be confused with bronchial asthma. The symptoms of this condition depend a great deal upon the severity of the infection but more so on the character of the exudate and edema.[10] The onset usually follows close upon a simple upper respiratory infection and often occurs very suddenly at night. There is respiratory difficulty, a characteristic brassy, barking cough, and the voice is hoarse or absent. Fever may or may not be a prominent factor. Asphyxia is present to a slight degree, and the pulse is usually rapid and regular. A consistent increase in the pulse rate indicates impending circulatory failure, and tracheotomy should be performed immediately instead of waiting until cyanosis occurs.

TREATMENT

Treatment of bronchial asthma may be divided into preventive, specific, and symptomatic.

Preventive Therapy.—

Since allergenic conditions, including bronchial asthma, are prevalent in children born of allergic parents, the following precautions may prevent or lessen the possibilities. Because it is impossible to avoid all inhalants and since infants and young children spend the greater part of each day in their bedroom, it is best to concentrate attention on that room. If the room contains more than one bed, all beds must be considered alike. There should be no feather pillows or down-filled covers in the room, and dust-proof covers should be used on bedding and pillows. The home should be as dust free as possible. Stuffed or fur toys should not be permitted; instead plastic, wood, or metal toys should be used. Floors should be bare except for washable cotton rugs; plain cotton window curtains should be used. Cotton blankets are usually warm enough; if woolen ones are necessary, they should be carefully covered and pinned down with cotton sheets. Cats, dogs, and canaries should be avoided. Talcum powder should not be used on the child, and the mother should not wear cosmetics containing orris root. The child should not be in the room when it is being cleaned and the windows are open.

A hot-air heating system is inadvisable, but if it is unavoidable, the system should have proper filters and a humidifier.

If an infant or child presents mild symptoms of allergic manifestations such as eczema, sniffles, recurrent bronchitis, or hay fever, it is advisable to have a prompt allergy survey made, including thorough skin testing. Contributory factors and infectious contacts and infections must definitely be avoided.

I personally believe that if these preventive measures were taken the incidence of bronchial asthma

would be greatly diminished in the older child and definitely diminished in the adult.

Specific Treatment.—

One method of specific therapy consists of complete elimination or the avoidance of allergens whenever possible, which, depending on their number, results in complete relief from symptoms with more or less a clinical cure.

The other specific method of therapy consists of desensitization. Allergens which cannot be avoided are prepared in extracts and given in injections of increasing amounts, usually over a period of months or even years. Results are usually good, but there is not a clinical cure.

A combination of avoidance and desensitization constitutes the best therapy.

Symptomatic Therapy.—

Since this paper is not a complete treatise on the diagnosis and treatment of bronchial asthma, I will discuss in part only those measures of practical importance for relief of an acute attack.

The patient should remain in bed. The room should be kept at an even temperature of about 72 F., fairly well ventilated, and the preventive measures previously mentioned should be taken. Smoking should be prohibited in the patient's room. The child usually feels much better in a semisitting position in bed supported by pillows or a chair.

The diet should be light and rich in carbohydrates; small amounts of sweetened fluids should be offered frequently. These fluids should be served at room temperature. Iced or cold foods should be avoided as they may aggravate or prolong an attack.

The patient must be given reassurance in order to avoid apprehension and anxiety on his part.

Aqueous epinephrine hydrochloride, 1:1,000, hypodermically is the drug of choice. The average dose is 0.1 to 0.3 cc. subcutaneously or intramuscularly. The best dosage is the smallest amount that will give the best results with the least side reactions. It may be repeated every 2 to 4 hours if necessary.

If prolonged effect is desired after the administration of the aqueous preparation, epinephrine in oil, 1:500, may be administered. The dose is 0.2 cc. to 0.5 cc., and it may be effective for as long as 12 to 16 hours.

Epinephrine, 1:100, may be effective as a pharyngeal spray; sometimes it affords relief within a few minutes. It must be sprayed deep in the pharynx during a deep inspiration. It is not necessary to compress the bulb of the nebulizer more than two or three times.

Ephedrine sulfate or ephedrine hydrochloride may be useful if no side effects occur. I prefer the hydrochloride since there is less tendency to gastric disturbance. It may be used as a nasal decongestant in 0.5 per cent strength to lessen mouth breathing. An ideal proprietary preparation which has a threefold action is NTZ. nasal solution which consists of Neo-synephrine, 0.5 per cent, Thenfadil hydrochloride, 0.1 per cent, and Zephiran chloride, 1:5,000. These components are a decongestant, an antihistaminic, and a wetting and antibacterial agent respectively.

Ephedrine only may be used in dosages from 8 to 25 mg. or ⅛ to ⅜ grain, but caution must be maintained to avoid overstimulation or narcosis. Combined with aminophylline, 30 to 60 mg. (½ to 1 grain), and phenobarbital, 7.5 to 15 mg. (⅛ to ¼ grain), it will aid in a mild attack and in the relief of cough (the dosages are for preschool age). It is administered three times a day if there are no side effects.

Aminophylline may be used to restore the normal reaction of epinephrine when it becomes ineffective after prolonged use. It is more effective rectally or intravenously. Rectally it is administered in the form of a suppository for the 4-to-5-year-old child, 3 grains per dose, repeated once or twice at 3- to 4-hour intervals. In the older child it is more effective intravenously in a dose of 1/20 grain per pound, not to exceed 4 grains. It must be given slowly and well diluted in a glucose solution (5 to 10 per cent), 150 to 200 cc. If vomiting or undesirable circulatory reaction occurs, the injection should be stopped and subsequent injections be smaller and slower.

Sedation may be necessary in the management of bronchial asthma to allay apprehension and provide the child with sufficient rest. Morphine should never be used. If sedation is necessary and must be immediate, paraldehyde diluted in olive oil and administered rectally is very effective and lasts for approximately 10 to 12 hours. Phenobarbital is quite safe and may be used in doses ranging from ¼ to 1 grain; its advantages are that it may be administered orally, rectally, or hypodermically. Antibacterial agents should be administered early in the attack if there is the possibility of an associated respiratory infection. The antibacterial agents may be penicillin, a broad-spectrum antibiotic, and/or a triple-sulfa preparation. It is wise to perform a sensitivity test and obtain a culture in order to determine the antibacterial agent of choice. The use of this one is then indicated in aborting subsequent attacks as a result of infection.

Syrup of ipecac is used for its expectorant effect and may offer considerable relief in infants and children. The dose is 3 to 4 minims every 3 to 4 hours during the first year and is increased by 1 drop for each year of age thereafter up to 1 cc. Although I never use syrup of ipecac to induce vomiting in order to loosen and expel the mucous plug in the bronchial tree, some physicians do; the dose for infants is 5 drops every 15 minutes until vomiting occurs. This dose is increased by 1 drop for each additional year. I have been fairly successful in stimulating the gag reflex with a tongue depressor; this avoids uncontrollable vomiting and subsequent acidosis.

Potassium iodide and syrup of hydriodic acid are used to liquefy the bronchial (secretions) mucus. The dose of potassium iodide is 1 grain per ounce of cough mixture; the dose of syrup of hydriodic acid is 1 part to 3 parts of cough mixture, preferably a fruit syrup. The dose is 1 teaspoon every 3 to 4 hours. If secretions become so abundant that cough is aggravated, the dose is decreased.

Antihistaminic drugs are particularly useful in the type of bronchial asthma due to pollens. Some patients respond to one antihistamine and not another. Benadryl and Pyribenzamine are administered in the dosage of 2 mg. per pound per day in four to six divided doses per day; the total dosage should not exceed 200 mg. The dosage of Chlor-Trimeton is 2 to 4 mg. two or four times a day.

Osteopathic manipulative therapy should be administered early in the attack, during the course of desensitization, and as a prophylactic measure to reduce attacks. It should be directed toward correction of any structural lesion in the cervical and upper dorsal region and ribs and in conjunction with soft tissue manipulation to avoid fatigue on the part of the patient. Treat-

ment should be repeated every 2 to 3 hours. The lymphatic pump should be used guardedly. In my experience the patient who receives osteopathic manipulative therapy has less respiratory difficulty and is less apprehensive.

If these measures are not satisfactory the patient should be hospitalized and treated for status asthmaticus. Reassurance should be given that the attack will be controlled by doing so.

The patient with severe dyspnea should be placed in an oxygen tent immediately instead of waiting until cyanosis occurs. The humidity of the oxygen tent should be kept relatively high to thin out and loosen the bronchial secretions. I have never had to resort to helium-oxygen mixtures in any of my cases. If necessary, it is best used in proportions of 80 per cent helium to 20 per cent oxygen so that the obstructed bronchi can be penetrated more easily.

Dehydration, which is usually the result of severe anorexia and/or vomiting and the resulting ketogenic acidosis, must be corrected. Hydration is corrected by intravenous administration of 250 to 500 cc. of 5 to 10 per cent glucose in water once or twice a day, depending on the age and weight of the patient and the amount of dehydration. This also assists in loosening bronchial secretions and in restoring liver glycogen. Aminophylline, sedatives (not morphine), antibiotics, and osteopathic manipulative therapy should be continued.

ACTH and cortisone are very effective and afford dramatic relief, but, nevertheless, their action is only temporary, and they have serious side effects. They are not substitutes for a complete allergic study and management.

The ACTH gel is considered the one of choice and usually only 3 to 5 days of therapy are necessary to terminate a severe attack. Antibacterial agents must be continued in full force in the presence of infection because the resistance to infection is greatly reduced during administration of these hormones. The recommended dosage for a child over 5 years of age is 20 to 40 units intramuscularly on the first day, 20 units the second day, 10 to 20 units the third day, and 10 units on the fourth and fifth days if necessary. For the younger child one-half or one-third of this dosage is recommended.

Oral administration of Cortisone is preferred. The recommended dosage is as follows for a child over 5 years of age: 25 mg. three or four times a day for the first day; 25 mg. twice a day on the second day; 25 mg. twice a day or if necessary on the third day; 25 mg. if necessary on the fourth day; 15 mg. if necessary on the fifth day.

When severe bronchial plugging is suspected or exists it is wise to administer 20 to 30 cc. of intravenous glucose in 25 or 50 per cent solution. However, bronchoscopic aspiration may be necessary to free the airway.

Tonsillectomy and adenoidectomy and removal of any foci of infection adjacent to the respiratory tract must be considered; the indications for removal are the same as in the nonasthmatic patient. If regrowth of lymphoid tissue follows, irradiation may help to lessen attacks. All elective surgery should be deferred until sensitivities have been controlled as far as possible.

The possibility of psychosomatic factors must not be overlooked, and, if present, they should be given proper attention.

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IMPROVED OUTLOOK FOR SURGICAL PATIENTS

Surgery is now undertaken with less risk to the patient than ever before. The mortality incidental to virtually all types of operations, even the most radical, has been materially reduced in recent years. A wide variety of factors have played a part in this achievement, particularly the better training of surgeons, advances in surgical techniques, and the wide use of the newer chemotherapeutic and antibiotic agents to control infection. Surgical patients have also benefited substantially from improved anesthetic substances and procedures, as well as from better preoperative preparation and postoperative care. Blood transfusion has been used increasingly to prevent death from surgical shock. The practice of getting patients out of bed as soon as possible after an operation has undoubtedly reduced the incidence of pulmonary embolism, which in the past not infrequently was the cause of sudden death following surgery. Furthermore, as surgery has become safer, it has become feasible not only to utilize more radical procedures but also to undertake operations in cases where surgery formerly would have been considered impracticable.—*Statistical Bulletin*, Metropolitan Life Insurance Company, November, 1954.

Journal A.O.A.
June, 1955

The Relationship of C-Reactive Protein and Antistreptolysin O Titer to Rheumatic Fever*

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INTRODUCTION

For many years investigators have attempted to devise some laboratory test which would definitely prove or disprove the diagnosis of rheumatic fever. This is particularly important when it is realized that the failure to diagnose and adequately treat rheumatic fever in the early stages increases the amount of crippling and the sequelae of the disease. To diagnose rheumatic fever in a child who does not have it results in unnecessary worry to the family and confinement of the child. The socioeconomic implications of both situations are obvious and need not be discussed at this time.

In the study of the cases reported in this paper, an attempt was made, where possible, to determine the sedimentation rates, to obtain electrocardiograms, and to perform pertinent x-ray studies. The history was carefully elicited and thorough physical examinations were performed; routine laboratory work included a complete blood count, serologic tests, and urinalysis. In addition, tests for C-reactive protein (CRP) and antistreptolysin O titers (AST) were performed.*

CASE PRESENTATIONS

Case 1. A 7-year-old white male was admitted to the hospital with a chief complaint of swollen, painful joints of 2 weeks' duration. His history revealed measles and a ruptured appendix with subdiaphragmatic abscess which required multiple blood transfusions 1 year prior to admission. There had been frequent sore throats with five paratonsillar abscesses which were lanced. The temperature was 104 F., pulse 120, and respiratory rate 40. X-ray examination revealed an enlarged heart and bilateral hypostatic bronchopneumonia. The electrocardiogram showed a PR interval of 0.14 second on the day of admission. Four days later the PR interval was 0.12 seconds. There was electrocardiographic evidence of myocardial damage. The sedimentation rate on admission was 35 mm. The patient was markedly anemic with 3,600,000 erythrocytes per cu. mm. and 9.2 grams of hemoglobin per 100 cc. The AST was positive in the 1:500 dilution and the CRP showed a 6-plus reaction. The diagnosis of acute rheumatic fever with cardiac failure and superimposed hypostatic bronchopneumonia was made on the basis of these findings. The patient responded well to treatment, and 10 days after admission the sedimentation rate was 25 mm. On the day of discharge, 12 days after admission, it was 15 mm. The AST was 1:250 and CRP was 3 plus.

Case 2. A 7-year-old white female was admitted with the chief complaint of recurrent abdominal pain which was not localized. The patient had had a similar attack 2 weeks prior to admission, and the chief complaint had been present for 24 hours. The temperature was 98.4 F., the pulse rate 88, and the respiratory rate 20. The sedimentation rate was 18 mm., and the leukocyte count 11,000 per cu. mm. with 8 per cent stab cells, 60 per cent segmented cells, 2 per cent eosinophils, 25 per cent lymphocytes, and 5 per cent monocytes. Because the pain and tenderness were not localized, the AST and CRP were determined in an attempt to rule out rheumatic fever. The AST was 1:50, and the CRP was 1 plus. The pain subsequently localized in the lower right quadrant. Operation was performed, and an acutely inflamed appendix was removed.

Case 3. A 9-year-old white male was admitted with a chief complaint of recurrent sore throat and chest pain of 2 weeks' duration. History revealed recurrent sore throat, measles, mumps, chickenpox, and two bouts of pneumonia. Tonsillectomy and adenoidectomy had been performed at the age of 4. The temperature was 102 F., the pulse rate 112, and the respiratory rate 36. An electrocardiogram revealed the PR interval to be 0.14 second. The sedimentation rate at the time of admission was 37 mm. An agglutination series for typhoid O and H, paratyphoid A and B, Brucella abortus, Pasteurella tularensis, Shigella, Proteus OXK, Proteus OX2, and Proteus OX19 was negative. Stool examinations were also negative. X-ray examination revealed atypical pneumonia of both hilar regions. The AST was positive in 1:125 dilution. The CRP was 2 plus. Examination of sputum which was produced after admission to the hospital revealed Hemophilus influenzae. The diagnosis was atypical pneumonia due to Hemophilus influenzae.

Case 4. A 5-year-old white female was admitted with chief complaints of sore throat, high temperature, and pain in the legs. A total leukocyte count was 12,600 per cu. mm. with 7 per cent stab cells, 82 per cent segmented cells, 2 per cent eosinophils, 25 per cent lymphocytes, and 4 per cent monocytes. The hemoglobin was 12 grams per 100 cc. The sedimentation rate was 54 mm. on admission and 4 days later had dropped to 32 mm. Throat culture revealed a hemolytic Staphylococcus albus infection. An agglutination series gave entirely negative results as did stool analysis and spinal fluid examination. Examination of the sternal bone marrow revealed it to be markedly hyperplastic. X-ray examination of the chest revealed chronic bronchitis. The AST and CRP reactions were negative. The patient responded well to antibiotics and was discharged on the sixth hospital day, cured of sore throat.

Case 5. A 13-year-old white female was admitted to the hospital with the complaints of hematuria, painful joints, and edema of the ankles and face. The total leukocyte count was 15,000 per cu. mm., with 12 per cent stab cells, 62 per cent segmented cells, 22 per cent lymphocytes, and 4 per cent monocytes. The sedimentation rate was 24 mm. The serum total protein was 5 grams per 100 cc. with 2.5 grams albumin and 2.5 grams of globulin. The AST and CRP were both negative. Urinalysis revealed an abundance of polymorphonuclear pus cells with clumps and an abundance of red blood cells. X-ray examinations were noncontributory. The patient was successfully treated for acute pyelonephritis.

Case 6. A 6-year-old white male was admitted to the hospital with chief complaints of cough and painful

*In the case presentations, C-reactive proteins are referred to as CRP and antistreptolysin O titers as AST.

joints of 2 weeks' duration. The temperature was 100 F., pulse rate 96, and respiratory rate 24. The sedimentation rate was 15 mm., and both CRP and AST were negative. The total leukocyte count was 14,000 per cu. mm. with 7 per cent stab cells, 60 per cent segmented cells, 23 per cent lymphocytes, 2 per cent eosinophils, and 8 per cent monocytes. The electrocardiogram was not abnormal. X-ray revealed bilateral unresolved bronchopneumonia. The patient was treated with x-ray and antibiotics and made an uneventful recovery. He was discharged 10 days after admission.

Case 7. A 5-year-old white female was admitted with chief complaints of cough and fever of a week's duration. The temperature was 100.6 F., pulse rate 112, and respiratory rate 32. The total leukocyte count was 12,500 per cu. mm. with 10 per cent stab cells, 55 per cent segmented cells, 30 per cent lymphocytes, of which 2 per cent were atypical, and 3 per cent monocytes. The AST was negative, and the CRP was 1 plus. The sedimentation rate was 9 mm. Analysis of the sputum resulted in positive cultures of Hemophilus influenzae. X-ray revealed atypical left upper lobe bronchopneumonia. Treatment for influenzal bronchopneumonia was successful.

Case 8. A 73-year-old white female was admitted with the chief complaints of nausea and abdominal pain which had been present for approximately 8 hours prior to admission. Her history revealed acute bouts of cholecystitis, numerous septic sore throats, heart disease, and thyrotoxicosis. She had undergone appendectomy at the age of 43 and thyroidectomy and cystocele repair at 67. Her temperature was 101.2 F., pulse rate 96, and respiratory rate 22. The sedimentation rate was 15 mm. The AST was 1:166 and CRP was 1 plus. Urinalysis revealed an occasional granular cast with abundant erythrocytes, and a 4-plus albumin. Electrocardiogram revealed tachycardia of the sinus type with a PR interval of 0.18 second. Hemoglobin was 13.6 grams with 4,320,000 erythrocytes per cu. mm. The total leukocyte count was 10,000 per cu. mm. The differential count was not remarkable. The patient was treated for acute pyelonephritis and was discharged in an improved state 5 days after admission.

Case 9. A 13-year-old white female was admitted to the hospital with the chief complaint of painful, swollen joints of the hands, arms, legs, and feet. Her history revealed numerous sore throats for the past 7 years, whooping cough, chickenpox, measles, and several bouts of rheumatic fever. The temperature was 99.8 F., pulse rate 88, and respiratory rate 16. The sedimentation rate was 22 mm. The CRP was negative, but the AST was positive in 1:500 dilution. The PR interval on the electrocardiogram was 0.16 second. X-ray examination revealed moderate bronchitis. Cultures from the throat contained hemolytic Staphylococcus albus. The diagnosis was acute rheumatic fever. The patient improved on a regimen of salicylates and penicillin and was discharged 10 days after admission.

Case 10. An 11-year-old white female was admitted to the hospital with chief complaints of joint pain and nosebleeds. Her history showed frequent colds, sore throats, bouts of nosebleeds, measles, and chickenpox. The temperature was 102 F., pulse rate 112, and respiratory rate 24. On admission she exhibited some cyanosis and dyspnea, marked edema of the lower extremities, and swelling of the joints of the hands. The PR interval on the electrocardiogram was 0.20 second. Sedimentation rate was 14 mm. The AST was positive in 1:500 dilution, and the CRP was 6 plus. X-ray examination of the chest revealed congestion and some pulmonary edema with the suggestion of early effusion. The cardiac silhouette was enlarged. There was a mitral systolic and a diastolic murmur. Cultures from the throat revealed Streptococcus pyogenes. The patient was placed on a regimen of salicylates with penicillin and was supported for 3 days with cortisone, 400 mg. per day. The patient showed steady improvement. On the fifth hospital day the sedimentation rate was 34 mm. She was discharged on the fourteenth hospital day, free of edema, joint pain, and dyspnea. The repeat AST and CRP were the same as on the day of admission. The sedimentation rate had dropped to 10 mm. The diagnosis was acute rheumatic fever.

Case 11. A 17-year-old white male was admitted to the hospital with the chief complaint of swollen ankles and feet and soreness of the joints of the left hand and elbow. These had been present for a week. A year prior to admission he had had a similar attack which was preceded by what was diagnosed as influenza. His history revealed measles, whooping cough, mumps, and frequent "streptococcal" sore throats. The temperature was 99.8 F., pulse rate 88, and respiratory rate 24. The sedimentation rate on admission was 27 mm. The PR interval of the electrocardiogram was 0.14 second. The T-waves were flattened. The CRP was 3 plus, and the AST was positive in 1:100 dilution. There was a systolic blowing murmur in the mitral valve sound area. X-ray examination revealed enlargement of the heart and increased bronchovascular markings in the lungs. The diagnosis was acute rheumatic fever. The patient was treated with salicylates and penicillin and was discharged 10 days after admission with complete remission of the chief complaints.

Case 12. A 38-year-old white male was admitted with the chief complaint of pain in the left hip joint and some dyspnea. Past history revealed St. Vitus' dance at the age of 10 or 12 years and transient paralysis. He had had frequent colds and sore throats as a child, but these had been infrequent in recent years. Hemorrhoidectomy had been performed a few years prior to admission. Physical examination revealed a harsh, mitral systolic and diastolic murmur over the apex of the left ventricle. The temperature was normal, pulse rate 64, and respiratory rate 16. X-ray examination revealed a prominence of the right atrial region with fullness in the region of the pulmonary conus on the left. Sedimentation rate was 2 mm. The PR interval was 0.24 second on the electrocardiogram. The AST was positive in 1:125 dilution, and the CRP was negative. The complete blood count was not abnormal. He was discharged with the diagnosis of inactive rheumatic fever and early mitral stenosis.

Case 13. A 59-year-old white male was admitted to the hospital with the chief complaint of black, tarry stools of 2 weeks' duration. The history revealed rectal surgery 7 years prior to admission, submucous resection 3 years prior to admission, and a stomach disorder of unknown type 3 years prior to admission which required frequent x-ray examinations. Cholecystectomy was performed 2 years prior to admission, and appendectomy at the age of 17. The physical examination was not revealing. The temperature, pulse, and respiratory rates were normal. The erythrocyte count was 3,300,000 per cu. mm. with a hemoglobin content of 10 grams per 100 cc. The AST was positive in 1:50 dilution, and the CRP was 1 plus. The sedimentation rate was 12 mm. Stool analysis revealed a 4-plus

reaction for blood. It was otherwise negative. Diagnosis was bleeding peptic ulcer. The patient was treated with an ulcer diet and responded well clinically; he was discharged from the hospital 8 days after admission.

Case 14. A 9-year-old white female was admitted with chief complaints of aches and pains in the extremities, upper right quadrant pain, and uncontrollable nosebleeds. Her history revealed frequent upper respiratory infections, several bouts of otitis media, fatigue, and anorexia of nearly a year's duration. Physical examination revealed a slight icteric tint to the scleras and an olive tint to the skin. The temperature was 101.6 F., pulse rate 116, and respiratory rate 24. There was a soft blowing systolic mitral murmur which was most pronounced at the anterior axillary line. The heart was markedly enlarged and presented a sinus bradycardia. The PR interval was 0.16 second. The sedimentation rate on admission was 20 mm. There was upper right quadrant pain and tenderness with enlargement of the liver for a distance of 4 cm. below the right costal arch. Hanger's cephalin-cholesterol flocculation was 4 plus at 24 hours. Thymol turbidity was 10. The AST was positive in 1:1,250 dilution, and the CRP was 6 plus. Diagnosis of acute rheumatic myocarditis and polyserositis was made. Treatment consisted of high doses of salicylates and penicillin. The AST 2 weeks later was positive in 1:500 dilution, and the CRP was still 6 plus.

Case 15. An 8-year-old white female was admitted with the chief complaint of headache, diarrhea, and vomiting of 4 days' duration. The temperature was 100.6 F., pulse rate 110, and respiratory rate 32. Clinical examination revealed an acutely inflamed mucous membrane of the pharynx and tonsils and moist crepitant rales at the base of both lungs. The total leukocyte count was 12,500 per cu. mm. with 8 per cent stab cells, 62 per cent segmented cells, 4 per cent eosinophils, 1 per cent basophils, 19 per cent lymphocytes, and 6 per cent monocytes. The hemoglobin was 12 grams per 100 cc. with 3,940,000 erythrocytes per cu. mm. X-ray examination revealed chronic bronchitis with some edema. Cultures of the throat were positive for Streptococcus salivarius (alpha hemolytic). The AST was positive in 1:12 dilution, and the CRP was 1 plus. The sedimentation rate was 10 mm. The patient was treated with triple sulfa and discharged 5 days after admission, clinically cured of streptococcal tonsillitis and pharyngitis.

Case 16. An 8-year-old white female was admitted to the hospital with chief complaints of aching arms and legs, nosebleeds, and extreme fatigue. She had had a cold and sore throat 2 weeks previously with severe tonsillitis and high fever. Her temperature was 103.2 F., pulse 110, and respiratory rate 28. The total leukocyte count was 12,000 per cu. mm. with 8 per cent stab cells, 59 per cent segmented cells, 2 per cent eosinophils, 27 per cent lymphocytes, and 4 per cent monocytes. The hemoglobin was 12.5 grams per 100 cc. The AST was positive in 1:1,250 dilution, the CRP was 6 plus, and the sedimentation rate was 38 mm. The PR interval on the electrocardiogram was 0.18 second. Throat culture showed a mixed flora which contained Hemophilus influenzae and a hemolytic strain of Staphylococcus albus. Other physical findings were soft systolic and diastolic murmurs at the apex which was in the anterior axillary line. Moist rales were heard over the entire chest. There was upper right quadrant tenderness, and the liver was palpable approximately 2 cm. below the right costal arch. The knees and ankles were swollen and tender to touch. The patient was treated with high doses of salicylates and penicillin and discharged after 2 weeks of hospitalization. Nose and throat cultures were negative. The AST at the end of 4 weeks was positive in 1:500 dilution, and the CRP was 4 plus. At this time the patient did not present any clinical symptoms of acute rheumatic fever.

Case 17. A 7-year-old white female was admitted to the hospital with the chief complaint of chills and fever of 2 days' duration. She also complained of a slight headache. Two weeks previously, she had had a sore throat with a high temperature but had recovered after 2 "shots" of penicillin. History revealed measles at the age of 5 with frequent sore throat. Physical examination revealed a soft blowing systolic murmur at the apex, left cardiac enlargement, and the apex almost at the anterior axillary line. The pulse was 110, temperature 102 F., and respiratory rate 36. The throat was bright red, and cultures revealed an infection with Streptococcus pyogenes. The sedimentation rate was 17 mm. The AST was positive in 1:500 dilution, and the CRP was 1 plus. Hanger's cephalin-cholesterol flocculation rate was 2 plus, and thymol turbidity was 6.5. The hemoglobin was 11.7 grams per 100 cc., and the total leukocyte count was 13,000 per cu. mm. with 7 per cent stab cells, 65 per cent segmented cells, 4 per cent eosinophils, 20 per cent lymphocytes, and 4 per cent monocytes. The patient was treated with salicylates and triple sulfa and discharged 1 week after admission in an improved condition and with a final diagnosis of acute rheumatic fever. The AST was positive in 1:500 dilution, and the CRP was 1 plus at that time.

Case 18. A 43-year-old white male was admitted with the chief complaint of upper right quadrant pain and dyspnea. There was a history of previous fat dyscrasia and alternate brown and clay-colored stools. There was also a history of excessive flatulence, particularly after eating such foods as cabbage. The temperature, pulse, and respiratory rates were normal. The complete blood count was not abnormal. The sedimentation rate was 12 mm. Results of gastrointestinal x-ray examination were not remarkable. The gallbladder series revealed multiple calculi within the gallbladder. Cardiac enlargement was suspected on physical examination although no murmurs were heard; x-ray examination revealed a slight left ventricular preponderance. The AST and CRP were negative. These were obtained because the attending physician suspected that the biliary calculi were not the entire cause of his complaints. The patient was operated on, and the gallbladder and calculi removed.

Case 19. An 11-year-old white female was admitted to the hospital with complaints of intermittent high fever, anorexia, dysphagia, and regurgitation of 2 weeks' duration. Two weeks prior to admission, she had had a sore throat which had been followed by the other symptoms. Physical examination revealed crepitant rales over the left chest, but there were no cardiac abnormalities either in size or valve sounds. The temperature was 101.2 F., pulse 116, and respiratory rate 36. X-ray examination of the chest revealed increased bronchovascular markings in the upper portion of the lower lobe and the entire upper lobe. Cultures of sputum revealed Streptococcus salivarius. The sedimentation rate was 11 mm. There were 15,000 leukocytes per cu. mm. with 22 per cent stab cells, 40 per cent segmented cells, 32 per cent lymphocytes, and 6

per cent monocytes. There were numerous atypical lymphocytes on the smear, and bone marrow examination was made to rule out possible blood dyscrasia. Bone marrow showed extreme hyperplasia of the normal elements. The test for infectious mononucleosis was negative. The AST was positive in 1 : 125 dilution, and the CRP was negative. The patient improved for a time on triple sulfas, but an exacerbation with spiking temperature occurred, and she was placed on a broad-spectrum antibiotic with good results. Cultures of the sputum and throat revealed Streptococcuc salivarius. The diagnosis was streptococcal bronchopneumonia. She was discharged in an improved state 2 weeks after admission.

TABLE I—SUMMARY OF CASES

Case	Age	Sex	AST	CRP	Sedimentation Rate in mm.	ECG-PR Interval in Seconds	Diagnosis
1	7	M	1:500	6 plus	35 15	0.12 to 0.14	Acute rheumatic fever with cardiac failure
2	7	F	1:50	1 plus	18		Acute appendicitis
3	9	M	1:125	2 plus	37	0.14	Hemophilus influenzae bronchopneumonia
4	5	F	Neg.	Neg.	54		Staphylococcus albus (hemolytic) infection of throat
5	13	F	Neg.	Neg.	24		Acute pyelonephritis
6	6	M	Neg.	Neg.	15		Bronchopneumonia
7	5	F	Neg.	1 plus	9		Hemophilus influenzae bronchopneumonia
8	73	F	1:166	1 plus	15	0.18	Acute pyelonephritis
9	13	F	1:500	Neg.	22	0.16	Acute rheumatic fever
10	11	F	1:500	6 plus	34	0.20	Acute rheumatic fever
11	17	M	1:100	3 plus	27	0.14	Acute rheumatic fever
12	38	M	1:125	Neg.	2	0.24	Inactive rheumatic fever and early mitral stenosis
13	59	M	1:50	1 plus	12		Bleeding peptic ulcer
14	9	F	1:1250	6 plus	20	0.16	Acute rheumatic myocarditis and polyserositis
15	8	F	1:12	1 plus	10		Streptococcus salivarius tonsillitis and pharyngitis
16	8	F	1:1250	6 plus	38	0.18	Acute rheumatic fever
17	7	F	1:500	1 plus	17		Acute rheumatic fever
18	43	M	Neg.	Neg.	12		Cholelithiasis
19	11	F	1:125	Neg.	11		Streptococcal bronchopneumonia
20	9	F	1:333	3 plus	35	0.14	Coronary insufficiency
21	40	M	1:100	Neg.			Collagen disease
22	42	M	Neg.	Neg.			Collagen disease
23	32	F	Neg.	Neg.	16		Rheumatoid arthritis
24	26	M	1:333	1 plus			Collagen disease
25	10	F	1:333	Neg.	18		Acute rheumatic fever
26	22	F	1:1250	6 plus	36		Acute rheumatic fever
27	65	M	1:125	3 plus	22	0.18	Rheumatoid arthritis
28	26	F	Neg.	Neg.	10		Collagen disease
29	31	F	Neg.	Neg.	21		Asthma and sinusitis
30	27	M	1:50	1 plus	6	0.14	Neurosis
31	18	F	1:500	3 plus	26	0.16	Acute rheumatic fever
32	16	M	1:500	4 plus	22	0.14 to 0.18	Acute rheumatic fever
33	17	F	1:500	Neg.	16		Streptococcic tonsillitis "type A"

Case 20. A 9-year-old white female was admitted with the chief complaint of chest pain and pain in the legs. It was particularly marked following slight exertion. There had been some edema of the lower extremities during the previous week. Past history revealed frequent nosebleeds and numerous upper respiratory infections. An electrocardiogram revealed marked myocardial anoxia with a PR interval of 0.14 second and depressed ST segments. The CRP was 3 plus, and the AST was positive in 1 : 333 dilution. Sedimentation rate was 35 mm. X-ray examination revealed left heart enlargement. Blood pressure was 120/60. There was a slight systolic murmur over the aortic region. The patient was discharged with the diagnosis of coronary insufficiency.

The remainder of the patients were outpatients who were not hospitalized although the patients in cases 25, 26, 31, and 32 had clinical acute rheumatic fever substantiated by laboratory findings. Case 27, a Jewish male, complained of painful swollen knee and ankle joints and when first seen had a positive AST in 1 : 125 dilution, a 3-plus CRP, sedimentation rate of 22 mm., and a PR interval on the electrocardiogram of 0.18 second. The AST and CRP were obtained again 2 weeks later, and at that time the AST was positive in 1 : 500 dilution and the CRP was 3 plus. In 6 weeks the AST was positive in 1 : 50 dilution, and the CRP was 1 plus.

DISCUSSION

Hemoglobin determinations were performed using the alkaline hematin method because of the rapidity with which they may be read in the colorimeter and the constancy of the reading. The amount of light absorption by the solution remains constant over a long period.

Erythrocyte counts were performed by diluting 20 cu. mm. of blood with 5 ml. of Gower's solution in a Klett test tube and mixing well before reading in the colorimeter. The method is that of Blum[1] and has several faults, namely, that in microcytosis the erythrocyte count by this method is too low and in macrocytosis the count is too high. However, its ease of performance and reproducibility far outweighs the disadvantage of occasional microcytosis or macrocytosis which can always be discovered by careful examination of the stained smear.

The sedimentation rates were performed by the method of Landau using the Landau-Adams microsedimentation apparatus. The normal values by this method are 1 to 6 mm. for children and adult males and 1 to 8 for adult females.[2] The sedimentation rate is increased whenever there is tissue destruction within the body. It is influenced by many factors, including the number of erythrocytes, blood proteins, lipoproteins, and electrolyte and water balance.[3-5] The sedimentation rates in all of the cases of acute or active rheumatic fever in this series were elevated.

The electrocardiograms were quite variable in the rheumatic fever patients. The cardiologists reported myocarditis in cases 1, 9, 10, and 18. In rheumatic inflammation of the heart, there is variation and prolongation of the PR interval on the electrocardiogram. The PR interval is usually 0.12 to 0.20 second normally, but occasionally may be normal at 0.24 second.[6] If there is doubt, lead II may be run with pressure on the carotid sinus followed by release of pressure.[7] If there is intrinsic disease of the heart as occurs in rheumatic fever, there will be no change in the length of the PR interval. In those patients with rheumatic fever on whom electrocardiograms were obtained, the PR interval by itself did not appear to be normal, but, when considered with the ventricular rate, it was deemed abnormal by the cardiologist.

It is interesting to note that in case 12 there was a prolonged PR interval which was probably due to myocardial fibrosis which followed rheumatic fever. One patient (case 27) with rheumatoid arthritis presented a PR interval of 0.18 second which was interpreted as indicating some myocardial disease. Baggenstoss and Rosenberg,[8] Bennett,[9] and Bayles[10] have reported and discussed the frequent occurrence of organic heart disease with the histologic features and behavior of rheumatic fever in patients with rheumatoid arthritis. We have observed these same pathologic lesions in our autopsy material. Case 27 illustrates the occurrence of rheumatoid joint changes and rheumatic heart disease.

The test for C-reactive protein had its origin in experimental testing for the somatic C-polysaccharide of the pneumococcus. This was reported in 1930.[11,12] This reaction was found to be nonspecific for the somatic C-polysaccharide,[13,14] as positive tests were also obtained in other acute inflammatory diseases such as rheumatic fever and osteomyelitis. The test has not been practical in the average clinical laboratory to date because of the difficulty in preparing the C-reactive protein antiserum. This material is now being produced commercially and is available under the trade name C.R.P.A. by Schiefflin & Company. The instructions for the performance of the test are enclosed with each vial of the C-reactive protein antiserum. The C-reactive protein which occurs in the acute phase of infections of human beings is found in the albumin fraction.[15]

The C-reactive protein is a nonspecific phenomenon, since such diseases as yellow fever, vaccinia, psittacosis, influenza, pneumonia, rheumatic fever, and streptococcal sore throat result in positive reactions with the specific C-reactive protein antiserum in the acute phase.[13] The C-reactive protein antiserum is presently prepared by reinjection of the purified C-reactive protein from acute phase serum into rabbits. The rabbits develop a high titer to this fraction in serum albumin and are then bled. The serum is separated and prepared for use by standardizing the titer. Originally, C-reactive protein was shown to be present by using the somatic C-polysaccharide substance to produce a precipitate with the acute phase serum. In their work, MacLeod and Avery[15] showed that only human beings and the higher primates are capable of producing C-reactive protein in their serum in response to infection.

The antistreptolysin titers of the patients reported in this paper were determined by the technic of Rantz and Randall.[16] The streptolysin O reagent is that manufactured by the Difco Laboratories who also supply the technic of the test together with the summary of the significant works concerned with antistreptolysin titers. We have modified the procedure by using a phosphate buffer with a hydrogen ion concentration of 6.5 to 6.7 in diluting the serum, and the reactions are more vivid and sharp. Originally, 0.85 per cent sodium chloride solution was used. Antistreptolysin titers were formerly obtained in this laboratory with streptolysin O reagent prepared after the method of Lancefield.[17] Dr. Lancefield of the Rockefeller Institute for Medical Research kindly supplied a strain of group A streptococcus which produces large amounts of streptolysin. The

preparation of the streptolysin O reagent was extremely difficult and required exacting technic. Bactostreptolysin O reagent produced by Difco Laboratories, Detroit, is a standardized streptolysin which has a fairly constant titer of reactivity and gives uniform results against a standard reference serum. The streptolysin O reagent is the lysin for erythrocytes which is liberated by a group A streptococcus. Numerous investigators have shown a high antistreptolysin titer in patients who have had recent acute infections with streptococci of group A.[18] Other investigators have found increased antistreptolysin titers in glomerulonephritis and the nephritides. The basic principle underlying the use of antistreptolysin titer is that rheumatic diseases are the result of infections with beta-hemolytic streptococci of the group A type.[19-21] There is always an interval of time between infection and the onset of clinical manifestations in rheumatic diseases. This is explained by the theory that any antigen-antibody reaction produces collagen diseases. Time is required to build up an antibody titer and for the antibody to combine with the antigen to produce the antigen-antibody complex which effects the anatomic change resulting in altered function and thus in the clinical manifestations of the rheumatic diseases.

SUMMARY AND CONCLUSIONS

From the series of cases presented here, it appears that the antistreptolysin titer (AST) and C-reactive protein (CRP) are helpful in confirming the presence or absence of acute rheumatic fever. AST titers of 1:166 or lower apparently are nonspecific for active rheumatic fever and indicate only previous infection by group A streptococcus. C-reactive protein reactions were of less intensity and magnitude in other infections, which were treated successfully with antibiotic or chemotherapeutic agents. In active rheumatic fever the CRP was always positive.

The most important factors in establishing the diagnosis of rheumatic fever are gleaned from the history and a careful physical examination of the patient. The findings include (1) history of an upper respiratory infection several weeks prior to the onset of symptoms, (2) joint pains and vague muscle aches of the legs and arms, (3) occasionally, abdominal pain which is sometimes localized in the upper right quadrant, in which case it is usually due to congestion of the liver, and (4) occasionally, dyspnea. Electrocardiograms may be variable but usually show changes in the PR interval. Leukocyte counts vary with the acuteness of the inflammatory condition. The sedimentation rates are usually elevated in proportion to the severity of the disease.

It is anticipated that there will be a plethora of reports on the use of C-reactive protein antiserum in the diagnosis of acute rheumatic fever as the material becomes more widely available and indications for its use become more recognized and better understood. The same is true of antistreptolysin titers.

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RESTRICTIVE DIETS

Although a restrictive diet is often an aid to treatment for disease, it is almost impossible for a patient to obtain exact amounts of specified foods daily over a prolonged period. Dietary restrictions for an acute emergency lasting only a few days, such as diabetic acidosis or a gastrointestinal upset, are easily tolerated, but if dietary restriction becomes a part of the treatment of a chronic disease numerous difficulties must be met. Because many physicians who prescribe such a diet do not appreciate this fact Rittelmeyer urges the physician never to prescribe a restrictive diet unless he has first tried it himself. . . .

Restrictive diets have been used in the treatment of obesity, gout, skin diseases, gallbladder diseases, nephritis, polycythemia vera, gastrointestinal diseases, and other conditions. If a psychosomatic element is present a difficult or unattractive diet may aggravate the emotional disturbance and thereby defeat the therapeutic regimen. If a restrictive diet must be prescribed, it is not enough to give the patient a list of foods he must avoid. He must be given verbal and explicit instructions regarding the method of preparation of the foods permitted, suggested menus, and instructions to be applied when eating away from home. Unless the physician is prepared to do this he would be wise to spare the patient the mental hazards of a restrictive diet.—Editorial, *Journal of the American Medical Association*, May 14, 1955.

Transcecal and Transappendicocecal Intubation of the Ileum

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Intestinal intubation is an important agent in the treatment of luminal obstructions. The Miller-Abbott tube or a variation has greatly enhanced the treatment of intestinal obstruction, reducing the mortality rate from 40 per cent to 11 per cent.[1-4] The use of a nasal tube for intubation is of most value in the treatment of paralytic ileus, but the use of this modality does not insure success. Difficulty in passage of the intestinal tube may be encountered in the following instances:

1. Because of lack of cooperation it is difficult to pass a Miller-Abbott tube in children and also in an irrational patient.

2. Distended loops of intestines may exert extrinsic pressure on the stomach, thereby inhibiting the passage of the tube through the pylorus.

3. In advanced cases of intestinal obstruction, reverse peristalsis may be of such magnitude as to prevent descent of the tube.

Valuable time may be lost attempting to pass a tube into the small intestines. As soon as the inability to pass the tube is recognized, operation must be performed in order to intubate the patient. In the past I have employed such classical measures as ileostomy or colostomy as the case warranted. Regardless of the procedure selected, I use Witzel's technic. This technic is applicable to any part of the intestine, although the outline which follows pertains to ileostomy.

1. The end of a Nélaton catheter is embedded in the antimesenteric border of the ileum pointing distally in the bowel. Interrupted Lembert sutures are taken in such a manner as to form a serosa-lined canal.

2. A retention suture is placed a short distance from the point of the proposed entrance into the lumen of the ileum.

3. A small opening is made into the ileum and the catheter is introduced into the ileal canal.

4. The end of the catheter is buried by using additional interrupted Lembert Sutures.

In addition to the above classical methods of operative intestinal intubation, I will describe two additional procedures that I have found useful in a certain number of selected cases.

1. Transcecal intubation of the ileum, in which case a tube extends through the cecum into the ileum, traversing the ileocolic valve.

2. Transappendicocecal intubation of the ileum, traversing the ileocolic valve.

I have performed transcecal intubation of the ileum in several instances with very gratifying results. The following case is a typical example:

I was called for consultation in the case of a patient with intestinal obstruction immediately following hysterectomy. A Miller-Abbott tube had been passed, but the obstruction persisted. Surgery revealed extensive adhesions due to inflammatory bands involving the terminal ileum. Lysis of adhesions necessitated the production of a large amount of raw surface of the serosal layer of the ileum. Reperitonealization of this raw surface was impossible, and transcecal intubation of the ileum was decided upon. The reasoning was as follows: a tube passed through the lumen of the ileum would not only aid in decompression, but would help maintain patency of the ileum in the area so susceptible to adhesions. Seven days postoperatively the tube was removed, and several days later the fistula closed of its own accord.

The following case is typical of those in which I have performed transappendicocecal intubation of the ileum: The patient was admitted with a ruptured appendix and marked adynamic obstruction. An attempt was made to pass a Miller-Abbott tube, but, since the patient was uncooperative, it was impossible. Surgery revealed generalized peritonitis with perforation of the appendix near its base. There was marked edema and friability of the base of the appendix and cecum. The small intestines were greatly dilated. Transappendicocecal intubation of the ileum was performed for the following reasons:

1. Intubation of the small intestines was an absolute necessity.

2. When an appendix perforates near the cecum, the incidence of a blowout is greatly increased, and internal or external fistula results. It was felt advisable to create a fistula rather than chance the formation of an internal fistula.

3. The presence of a tube in the lumen of the ileum would aid in the maintenance of its patency.

The technic for transcecal intubation of the ileum is as follows:

1. Rubber clamps are placed on the colon superior to the cecum, and a rubber clamp is placed on the ileum 6 inches from the ileocolic valve.

2. A small incision is made on the anterolateral wall of the cecum, and a catheter is threaded through the cecum into the ileum, traversing the ileocolic valve. It may be difficult to traverse the ileocolic valve because the ileum projects into the cecum as two folds.[5] The catheter may be guided through a trocar.

3. The catheter is threaded approximately 6 inches into the ileum and is then fixed in place in the cecum in a manner similar to Witzel's technic.

4. The blunt end of the catheter is then brought out through a stab wound, and usually a suture is taken from the adjacent anterior parietal peritoneum to the cecum.

5. The catheter end may be connected to suction or gravity apparatus and frequent irrigation performed. Transappendicocecal intubation of the ileum is carried out in a similar fashion, except that the catheter enters the base of the appendix rather than the cecal wall.

SUMMARY

A brief review of current methods of intestinal intubation has been given, and it has been emphasized that the procedure must be selected according to the particular problem encountered.

Two technics have been described, transcecal and transappendicocecal intubation of the ileum. Two cases have been cited which show the usefulness of the procedures. I have used these technics over a period of 8 years and, to date, have been very gratified with the results.

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Lower Nephron Nephrosis*

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Acute renal insufficiency, or lower nephron nephrosis, is a constant threat to every patient who undergoes a surgical or an obstetric procedure or is exposed to shock, whether it is hemorrhagic, antigenic, or traumatic. It is the obligation of every practitioner to be constantly on the lookout for the early symptoms of this syndrome so that a physiologic approach to therapy may be commenced and thereby afford the patient as high a survival chance as possible. This paper will deal for the most part with the early diagnosis of the syndrome of lower nephron nephrosis and a physiologic approach to its therapy.

PATHOGENESIS

The pathogenesis of the syndrome has yet to be fully established. Every patient who has some kidney dysfunction has a greater chance of developing acute renal insufficiency than do patients with normal kidneys. It is thought that chronic or subclinical renal insufficiency has a direct bearing not only on the development but also on the prognosis of acute renal failure. It therefore behooves the physician, whenever possible, to evaluate fully the renal status of his patient before subjecting him to any traumatic situation.

The most widely accepted theory of the pathogenesis of this syndrome is that it develops from shock, usually acute. The idea that prolonged subclinical shock may precipitate the same situation as a shorter period of acute shock is coming into prominence. The seat of the pathologic changes is in the kidneys. The pathophysiologic changes occurring in the renal arterioles to combat the shock state are due to constriction; the length of time that this state of constriction continues varies with the severity of the shock. The renal tubules (distal convoluted and loop of Henle mainly) are extremely sensitive to the anoxic state that follows arteriolar constriction, and they undergo certain pathologic changes that will be described briefly below. Another popular concept of the pathogenesis of lower nephron nephrosis, especially that associated with the transfusion reaction, is that of hemolysis of the erythrocytes with incompatible blood and the formation of heme casts in the tubules. It has been shown by Yuile[1] that heme crystals may be formed by tubular necrosis itself without the presence of a transfusion reaction in experimental animals. It has also been proved that alkalinization of the urine will not prohibit heme formation and that heme crystals are as apt to form in an acid medium as in an alkaline medium. Although heme casts are important as far as furthering renal damage is concerned, they play little or no part in the precipitation of renal shutdown.

*Presented at the Eastern Study Conference of the American College of Osteopathic Internists, Philadelphia, March 12, 1954.

ETIOLOGY

A partial list of etiologic factors includes: severe hypotension, hemoconcentration, nephrotoxic substances from injured tissues, liver failure (no deaminization and its effect on sodium and water metabolism), transfusion reaction, transurethral resections (it has been reported that as many as 4 per cent of patients develop reversible renal failure after transurethral prostatectomy,[2] blackwater fever, favism, poisoning (sulfa, plasmochin), burns, crush syndrome, Haff disease, eclampsia, and hemorrhage. It is also thought that prolonged use of pressor substances such as Levophed can contribute to the pathogenesis of this syndrome. It can definitely be stated that of patients who have been through a period of shock and show signs of sympathetic hyperactivity (such as pallid clammy skin with a rise in blood pressure of 20 to 60 points above the normal level for that patient) about 10 per cent will pass into an oliguric or anuric state.

Probably the most complete description of the micropathology of this syndrome is that of Lucké.[3] McManus[4] states that there are five definite features seen in the kidney with any fixation and stain in lower nephron nephrosis: (1) Normal glomeruli, (2) flattened tubular epithelium, (3) absence of colloid droplets in the epithelium of the proximal convoluted tubules, (4) casts in tubules, including pigment casts, and (5) tubulovenous thromboses. As is evident, there is involvement of the entire nephron to some degree, and from this involvement comes the term "lower nephron nephrosis."

PROGNOSIS

It is thought that with complete renal shutdown the average patient can survive about 3 weeks. The longest reported survival is 7 weeks.[5] Experimentally it has been shown that survival time seems to vary with the size of the animal. The smaller the animal, the longer the survival and vice versa. Lucké,[3] in his studies, arrived at the following conclusions after studying 100 patients with renal failure: 95 per cent of these patients died within 14 days after the onset of either oliguria or anuria; 86 per cent within 10 days and 48 per cent within 6 days. He had no deaths in patients who survived more than 20 days. It can be assumed from these statistics, and it is verified clinically, that the longer the patient survives the better is his chance of ultimate recovery. The critical period following renal shutdown as a result of hemolytic reactions lasts approximately 2 weeks. If a patient can survive this period he has an excellent chance not only of living but of a complete return of kidney function. Since it is almost impossible to differentiate between moderate, mild, and severe kidney damage, by ignoring the se-

verest type it can be stated statistically that about 66 per cent of patients (mild to moderate) recover under conservative management; about 20 per cent die of other things such as massive pulmonary hemorrhage and embolism—strangely, fat embolism is not uncommon in this situation—and about 10 per cent of all these people die of unrelenting renal failure.[6] Prognostically and therapeutically it seems that the better the fluid and electrolytic balance maintained during the oliguric or anuric phase, the better is the over-all prognosis. Finally, as is logical, the oliguric patient has a better chance for recovery than has the anuric patient.

DIAGNOSIS

One would think lower nephron nephrosis would be easy to diagnose, but quite to the contrary, the diminishing urinary output causes diagnostic confusion. It is imperative in this syndrome that the diagnosis be established as early as possible so that an overzealous physician will not force fluids into his patient, precipitate pulmonary edema, and drown the patient in an attempt to force kidney function. Lower nephron nephrosis should be suspected in every patient who has been through a period of shock following which the urinary output is 400 cc. or less in 24 hours. The urine should be examined and its specific gravity obtained and the phenolsulfonphthalein, serum creatinine concentration, and benzidine tests should be performed early in any case of oliguria with suspected renal failure.

In lower nephron nephrosis physical examination of the urine reveals a rather fixed specific gravity of 1.012 or 1.010. Usually proteinuria is present, whereas it did not exist before. The urine has the appearance of cold grapefruit juice. These findings are not diagnostic but are highly significant in building a diagnostic platform.

The phenolsulfonphthalein test is probably the best diagnostic aid. This test is the easiest to perform, and there is the least chance for technical error. The phenolsulfonphthalein test characteristically indicates a trace in any urinary specimen obtained, even those collected after 1 or 2 hours. It is of particular importance when oliguria is coupled with a high specific gravity. Specific gravity above 1.030 indicates dehydration, but in cases of lower specific gravity, especially in the presence of proteinuria, the phenolsulfonphthalein test will indicate whether the condition is reversible renal failure or simple dehydration. A relatively normal excretion can be expected with dehydration. Lyon[2] states ". . . that within 48 post-shock hours, no cases of reversible failure have excreted more than a trace of PSP in the half-hour specimens." This adds further to the diagnostic value of this procedure.

Another laboratory test that can be utilized in making this diagnosis is the serum creatinine concentration. This end product of muscle metabolism is the most sensitive serum indicator of sudden renal failure. The serum creatinine may double to triple its normal concentration within the first 24 hours of kidney failure; the nonprotein nitrogen increase may be no greater than would be expected with marked dehydration, thus making serum creatinine a much more sensitive and earlier diagnostic aid.

Finally, the benzidine test for hemoglobinuria is almost always positive (denoting heme crystals in the urine). This usually is seen in lower nephron nephrosis associated with the transfusion reaction.

Before starting a discussion of the treatment of lower nephron nephrosis, a brief resume of the necessary laboratory procedures in the management of this syndrome is indicated.

1. An erythrocyte count should be made. Every practitioner knows of the necessity of oxygen for metabolism. Here it is essential to maintain an adequate oxygen-carrying power in the blood. If anemia is present, whole blood transfusions should be used very cautiously. Whole blood transfusions not only increase the blood volume and increase the load on the heart but introduce more of the end products of metabolism of which there are already too many present in the blood. The most logical answer is the use of washed red blood cells, transfused in small quantities over a period of a few days. This will increase the oxygen-carrying power of the blood and very little else.

2. Blood urea nitrogen should be determined at least every other day as it is an invaluable aid to the effectiveness of therapy. It is not a prognosticator since the blood urea nitrogen finding has little or no relation to the severity of the renal damage.

3. Urinalysis should be performed daily in the oliguric patient. Urinary chloride determination will help in maintaining electrolytic balance, and this may be determined many times a day. Since the hydrogen ion concentration of the urine is a reflection of the acid or alkaline tendency of the plasma, it should be watched very carefully. If the hydrogen ion concentration is between 5.0 and 7.0 it is most probable that anions and cations are being lost in the urine proportionately. If the hydrogen ion concentration is less than 5.0, the loss of chloride is greater than that of sodium or other cations. If the hydrogen ion concentration is 7.5 or more, more cations than anions are being lost in the urine, a condition that can easily lead to acidosis.[7] Specific gravity is fixed in this syndrome; changes denote improvement in the patient's prognosis and a return of some tubular function.

4. Plasma proteins and albumin-globulin ratio should be determined frequently so that these important osmotic and metabolic factors may be maintained. Hypoproteinemia certainly does not help an already embarrassed glomerulus to function. Also, the buffering effect of the plasma proteins must be remembered in the fight against fatal acidosis.

5. The serum creatinine concentration should frequently be determined since its improvement immediately betters the prognosis.

6. Blood chlorides, calcium, sodium, potassium, carbon dioxide-combining power, and the hydrogen ion concentration of the blood should be determined when the diagnosis is established and repeated when indicated.

7. Electrocardiograms should be made every few days for further insight into the status of potassium metabolism and the general cardiac status.

8. Blood volume determination, though done infrequently, is probably the best way of estimating fluid balance. This, compared with fluid intake and output and change in body weight, should leave little or no doubt as to the fluid needs of the patient.

TREATMENT

The treatment of lower nephron nephrosis should be a well-modulated middle-of-the-road program between very active therapy and watchful waiting. The success of therapy can be no better than the quality and proficiency of the laboratory, whose findings will dictate a great part of the treatment in this syndrome.

Period of Shock.—

The most active part of treatment begins with the first phase of this triad, namely, the period of shock. Shock, the most common etiologic agent, must be attacked vigorously with antihypotensive agents, plasma, and plasma expanders. Whole blood transfusions should be used with caution. They are contraindicated if the etiologic factor is a transfusion reaction. The other adjuncts for the treatment of shock (stimulants, heat, et cetera) should be used at the discretion of the physician in charge.

As an osteopathic physician, I think that this would be a good place to pause and consider the role of osteopathic manipulative therapy in this syndrome. The use of osteopathic manipulative therapy begins with the first phase as an integral part of the treatment of shock. Stimulation of the upper dorsal regions will enhance respiratory functions and will cause some elevation of the blood pressure. This stimulatory technic should be carried on intermittently during the whole shock period. In the oliguric phase, the role of osteopathic manipulation comes to the fore. In this stage, attention must be directed to the renal splanchnics, and soft tissue manipulation should be given for 5 minutes at least every 2 hours. In the diuretic and recovery phase osteopathic manipulative therapy, if given regularly, will speed the return of kidney function to normal.

Oliguric Phase.—

Before discussing the treatment of the oliguric phase it is best to consider first the objectives of therapy:

1. Maintenance of electrolytic balance
2. Maintenance of fluid balance
3. Provision for caloric needs
4. Decrease of the metabolic load on the kidneys and body as a whole
5. Maintenance of life till kidney function returns.

As stated above, the nearer to normal hemostasis a patient is maintained, the more favorable is the prognosis, the easier is the treatment during the diuretic phase, and the more complete is the return to normal kidney function with less residual renal damage.

It has been well established that during the oliguric phase the fluid losses are: (1) Urinary output plus (2) loss via lungs (400 cc./24 hrs.) plus (3) loss via skin (600 cc./24 hrs.) plus (4) loss via feces (150 cc./24 hrs.) plus (5) an extension above these basic amounts for fever with perspiration, vomiting, diarrhea, et cetera. Since it is known that kidney damage is present and that renal function is almost nil, it certainly is not logical or physiologic to force fluids intravenously or "pry" open the kidneys and force diuresis. The objective is to replace fluids which are lost and keep as close a physiologic balance as possible. From the above figures it can be seen that the basic requirement is a mimimum of 1,150 cc. It is rare in a case of lower nephron nephrosis with its concomitant uremia to maintain this physiologic balance via the oral route. In planning a therapeutic approach it is best to be ready to administer everything by the parenteral route.

To maintain a proper caloric balance in a patient it is best to use intravenous glucose. It also helps to reduce the hyperpotassemia that may be present through its storage as glycogen. Furthermore, it is a good protein sparer, and this will help to reduce further the production of nitrogenous wastes and attain proper nitrogen balance. It has been established that the average patient in bed needs about 1,000 calories per day. These may be given in combination with the fluid replacement; if 10 per cent glucose solution is used, 400 calories can be administered in every 1,000 cc. of fluid. Any deficit in caloric needs may be given in a 50 per cent solution of glucose. This gives 100 calories in every 50 cc. of fluids administered. Some diuretic effect will be obtained from this and the caloric requirements of the patient will be supplied.

Hyponatremia and hypochloremia develop during the oliguric phase along with hyperkalemia and hypocalcemia. Low sodium and chlorides in the blood are very easily induced by the physician who, in his attempts to force diuresis through the intravenous use of fluids, dilutes the electrolytic content of the blood. It has been shown recently that during a state of shock, potassium leaves the cells and enters the blood and interstitial compartments.[7] To balance this, sodium and chloride enter the cells and this is the beginning of a depleted sodium reserve. A shift of water from the cellular elements to the interstitial spaces also carries some sodium and chloride with it. Failure of the kidney to save electrolytes results in further loss of sodium and chloride, although the amount is small in the oliguric patient. Probably the most important loss of sodium and chloride is in the buffering system of the blood. Here sodium and chloride both are tied up by the acidic elements of the blood (due to uremia) in order to compensate for the acidosis that is present and maintain the hydrogen ion concentration of the blood.

Bland[7] says that if the patient doesn't perspire too much, the water intake should be 1,500 ml. containing 50 mEq. each of sodium and chloride in order to maintain these components in their proper proportions. (The amount secreted in the urine is to be added to this.) If the patient perspires enough to necessitate a change of sheets or pajamas, then 2,000 ml. of a solution containing 75 mEq. (plus urinary excretion) each of sodium and chloride is needed. This method is very empirical but may be followed if frequent sodium and chloride blood levels cannot be determined. If these blood levels can be determined daily, the sodium and chloride deficit should be added to the daily fluid needs. If this is not feasible, chlorides alone may be given by the addition of ammonium chloride. This will introduce more nitrogen into the circulation, but the effects of a low chloride level are more deleterious than the rising nitrogen levels.

The sudden and rapid deaths during the oliguric phase of this syndrome are undoubtedly due to hyperkalemia. The fatal effect of hyperkalemia need not be discussed here, only the mechanism of its formation and its treatment. It must be remembered that potassium is an integral part of every cell and is the basis for many cellular functions. Changes in the blood potassium levels affect the body functions profoundly. Potassium is affected by adrenal hormones, pituitary hormones, and the general electrolytic state of the body. During the oliguric phase of renal failure, the epithelium acts as a dead membrane, and any potassium excreted by a functioning glomerulus passes back across the tubular membrane into the blood instead of on to the bladder. Therefore, it may be stated that there is acute retention of potassium during the second stage. Normally, there are from 10 to 100 mg. of po-

tassium excreted by the kidneys per day. Naturally, it follows in renal failure that this amount from normal metabolism is retained in the blood and extracellular compartments. Furthermore, since acute renal failure is often seen as a result of incompatible blood transfusions, hemolysis of the red blood cells frees potassium into the general circulation.

In the presence of cellular anoxia there is also a shift from cellular to extracellular compartments by the potassium ion. As stated above, adrenal hormones, intimately concerned with the shock state, increase the circulating potassium levels. The metabolism of 1 gram of protein liberates 2.4 grams of potassium. Finally, glucogenesis from stored glycogen results from the breakdown of potassium-hexose-diphosphate, liberating still more potassium that cannot be excreted or stored efficiently by the body. It is a wonder that all of these patients do not succumb to the deleterious effects of hyperkalemia when these factors are added to a non-excreting kidney and the multiple exogenous sources of potassium.

The most accurate method of obtaining the blood levels of potassium is with the flame photometer. Since this is not universally available, daily electrocardiograms should be made with special attention being paid to the Q-T interval, the S-T segments, and the T wave duration. Change in these intervals is very significant and will give a great deal of information concerning potassium levels and general electrolytic balance.

With the earliest signs of renal failure all exogenous sources of potassium should be deleted from the diet and therapeutic regimen. Intravenous fluids should be free from potassium in any form. This excludes the use of Darrow's solution, Ringer's solution, and Ringer's lactate which are the common replacement fluids. After establishment of hyperkalemia, the artificial kidney, peritoneal lavage, gastric lavage, and exchange resins may be used. A physiologic way of decreasing extracellular potassium is by administering glucose and insulin. As stated above, potassium is intimately related to the storage of glycogen and glycogenesis. This fact is proved time and time again by the occurrence of hypopotassemia during the treatment of diabetic acidosis and coma with large doses of insulin.[7] Many patients show a disappearance of electrocardiographic signs of potassium imbalance when treated with intravenous infusions of glucose and insulin. Not only is the use of glucose and insulin a safe and judicious form of therapy, but it also helps supply the caloric needs of the patient. Glucose may be given as a 25 per cent solution with about 1 unit of crystalline insulin for every 2 grams of glucose used.[7] One author[2] recommends the use of a 50 per cent solution of glucose in a steady drip supplying 2,500 to 3,000 calories per day, and he has reported reduction of plasma potassium levels from 7.0 to 5.5 within 24 hours. This is a more radical approach to the problem and necessitates using a greater volume of fluid and will damage the veins used.

Hypertonic saline solution plays an important role in the correction of electrolytic imbalance. Not only will it help correct this imbalance, but it helps restore plasma volume. With an excess of sodium present, the potassium will be forced back into the cell. Since sodium and potassium are physiologic antagonists and excess sodium is the lesser of two evils, it would be better to have an excess of the former than the latter. It might be well to mention that sodium may be given as sodium bicarbonate (a 5 per cent solution) or sodium chloride (same percentage), the former being very useful in returning the carbon dioxide-combining power of the blood to normal. Since many cases of renal failure are reversible and the patient's life can be saved, it is of great importance that he not die of hyperkalemia during the oliguric phase.

Another type of therapy for hyperpotassemia is the dilution method. This consists of giving intravenous fluids until edema occurs, thus diluting the potassium. This may be accepted for control of hyperpotassemia, but it certainly overloads the circulation and adds a great strain to an overloaded heart.

Little has been published to date regarding the status of calcium metabolism as related to renal insufficiency. It has been established that in diseases of the kidney the renal epithelium loses the ability to conserve basic ions by virtue of its inability to synthesize ammonium radicals. Furthermore, calcium and its salts are not reabsorbed from the glomerular filtrate when the renal tubule becomes a dead membrane in lower nephron nephrosis. In this syndrome calcium is excreted in relatively large amounts, depending on the degree of oliguria. Since there is no exogenous source of calcium ion in most of these patients, there is a very good possibility of the symptoms of hypocalcemia developing at the beginning of the diuretic phase. There is also hinted in the literature a relationship between potassium and calcium metabolism.[7,8] It is known that hypokalemia will mask the tetanic manifestations of hypocalcemia. In the diuretic phase, as the lost potassium is replaced, there is a good chance that tetany may ensue. It therefore becomes necessary to ascertain the level of blood calcium during the oliguric phase so that the available calcium will not be depleted and so that the calcium-phosphorus ratio will not be significantly altered. Calcium is easily administered in the form of the gluconate and is given intravenously as needed. It may be argued that intravenous calcium is contraindicated in the presence of a digitalized patient, and many of these patients require digitalis. In the Hospital of the Philadelphia College of Osteopathy, however, intravenous calcium has been used in at least fifty digitalized patients with no deleterious effects. If given cautiously and, above all, slowly there will be no adverse reactions to it.

No discussion of the oliguric phase would be complete without mention of a few of the modalities that have been used in the past and still are being used as part of the treatment of this syndrome.

Drug Therapy:

1. Heparin. Early use of anticoagulant therapy in lower nephron nephrosis has been advocated by some. They feel that there are definite thromboses in the renal arterioles and that anticoagulants will dissolve these and re-establish renal continuity. Caution should be used with these drugs, especially since most of the patients are in a postoperative period, and the danger of postoperative hemorrhage must not be underestimated.

2. Testosterone (25 to 50 mg. intramuscularly daily). The action of this steroid is thought to be anabolic at the cellular level. It causes retention of sodium. It enhances and increases protein synthesis and so decreases the amount of nitrogenous metabolites. It gives the patient a sense of well-being and so makes him more cooperative.

3. Exchange resins. These are being used more frequently to decrease hyperkalemia. Since the gastric juices are rich in potassium, carboresins combine with

these ions and are excreted via the lower gastrointestinal tract. They absorb almost as much and possibly more sodium than potassium, are difficult to take, and may produce gastrointestinal upsets. They may be given as retention enemas, which may be very effectual.

4. Digitalis. This drug is very useful in the treatment of lower nephron nephrosis. If used correctly it will save a patient's life until the kidneys begin to function. A full digitalizing dose should be given intravenously, probably over a 24-hour period, at the first signs of congestive failure. The patient must not be put on a routine maintenance dose since the excretory mechanisms for digitalis are not functioning correctly. It is a very simple thing to create digitalis poisoning in these patients. They should be maintained according to the clinical symptomatology.

5. Mercurial diuretics are contraindicated in acute renal failure.

6. Antibiotic therapy is useful in the prevention of secondary infection and should be used in every case.

7. Vitamin therapy should be used as indicated.

Physical Modalities:

1. Gastric lavage. This method of treatment is very successful in uremia and hyperkalemia. A Miller-Abbott tube is passed to about 5 feet beyond the pylorus and attached to a continuous suction apparatus. A Levin tube is passed into the stomach. Through the Levin tube is passed physiologic saline, M/6 lactate as a solution containing: sodium chloride, 6 grams per liter; sodium bicarbonate, 3 grams per liter; calcium chloride, 0.1 gram per liter; magnesium chloride, 0.1 gram per liter; and glucose, 20 grams per liter. This solution is passed through the Levin tube at the rate of 10 liters per 24 hours. It is reported that 3 grams of urea and as much as 1.2 grams of potassium are removed from the blood stream in 24 hours.[9] This form of therapy is easily managed and is a successful antidote for toxicity.

2. Peritoneal lavage. The purpose of peritoneal lavage is the same as that of intestinal lavage. Since it requires surgical incisions, heparinization, and is frequently accompanied by peritonitis, its side effects and its lack of effect in combating toxicity rule it out of therapy.

3. Caudal, spinal, and paravertebral anesthesia. These may be tried in order to enhance renal flow in the undamaged nephrons. The results are not too spectacular.

4. Stupes, high colonics, heat, diathermy. These all have been used in treatment of this syndrome. In the average case they do nothing more than warm the skin and clean out the bowel. They do little or nothing toward reversing the renal insufficiency or correcting the electrolytic imbalance.

5. Artificial kidney. Researchers have for many years attempted to develop an apparatus that can take over the function of the kidneys and so put them to rest. Elkinton and his associates[10] at the University of Pennsylvania have developed a machine that has been very successful and which operates something like this: The patient is heparinized, and a convenient artery and vein are cannulized. The blood, under artificial pressure, is pumped through coils of cellulose acetate tubing wound tightly around a rotating drum. This revolves through a bath of 100 liters of a fluid containing: 0.6 per cent sodium chloride, 0.2 per cent sodium bicarbonate, 0.4 per cent potassium chloride, and 1.5 per cent glucose. Dialysis can be controlled by changing the fluid in the bath according to the patient's electrolytic needs. Proteins pass this membrane poorly and with difficulty, so little or none is lost. This form of therapy loses its effectiveness in inverse proportion to the duration of oliguria. Its main purpose is to keep the patient alive until the function of the kidneys is re-established. If started early, it will do its job well.

In summarizing the oliguric phase it can be said that the closer the patient is maintained to normal electrolytic and fluid balance the greater are his chances for successfully passing through the diuretic phase and going on to complete recovery.

Diuretic Phase.—

The onset of the recovery period in lower nephron nephrosis is heralded by massive diuresis. This will start unexpectedly with no apparent change in the condition of the patient. The patient may start the day by voiding the expected few cubic centimeters of urine and may urinate a few thousand cubic centimeters by the end of the day. With the onset of diuresis, the chances of the patient's survival are theoretically greatly enhanced. This does not mean that with diuresis the crisis is over and the patient will live. Quite to the contrary, the patient has moved from one morbid situation into another, perhaps even more complex than the first. The outcome of this third stage of lower nephron nephrosis depends to a great extent on the physician and his ability to maintain the patient's physiologic balance during it. The greater the excess of electrolytes and fluids, the greater will be the loss. Furthermore, if there is a deficiency state at the onset of diuresis, it follows that the chances of survival are greatly diminished.

The diuretic phase may begin from the second to the twelfth day; in a few cases on the fourteenth to the sixteenth day and in some as late as 2 to 3 weeks. Although diuresis may occur from the second to twelfth day, physiologic restitution may take many months. With the regeneration of the tubular cells, the kidney no longer urinates into itself but begins to pass urine down into the bladder. The ability to concentrate is the last physiologic function of the kidney to return to normal. As is expected, the specific gravity of the urine is very low and is fixed. As much as 7 or 8 liters of urine may be passed during a 24-hour period, and this amount may be continued for from 4 to 7 days. During this time it is of importance to replace a large percentage of this fluid. From 3,000 to 5,000 cc. in 24 hours may have to be given to help replace this loss. The intravenous route should be dispensed with at the earliest possible time.

The urine excreted during the diuretic phase is practically the same as the glomerular filtrate, so that there is no change in the fluid that passes the glomerular membrane, which, by the way, is damaged and permits the protein molecule to escape, or in the fluid in the bladder. There is no reabsorption at the tubular level. As can be expected, all electrolytes are lost from the circulation in proportion to their concentration, and blood levels are constantly changing.

Sodium and chloride are lost in great quantities. This continues not only during the diuretic phase but may continue well into the convalescent period causing

a recently recognized condition, the salt depletion syndrome. Although there are no definite figures regarding the loss of sodium and chloride, many grams are lost a day. This loss may be roughly computed at the bedside by using Bryant's modification of the Fantus test (potassium chromate, 10 per cent, plus silver nitrate, 0.73 per cent). Sodium chloride may be replaced on the basis of chlorides excreted per liter of urine. This test may have to be done every few hours because of the fluctuations in the blood levels. It is also advisable to determine blood chlorides and sodium levels as a further check if possible. Sodium chloride may be given intravenously or orally, whichever is more feasible.

Blood levels of potassium once again are a major stumbling block in therapy. There is as much likelihood for the patient to die from hypokalemia as from hyperkalemia, and these levels again must be watched very carefully. Since it is inevitable that the patient will have some degree of hyperkalemia, it follows that there will be an excess loss of potassium during the stage of diuresis. Potassium is excreted in the urine in amounts of about 500 mg. per liter. Since 5 to 6 liters of urine may be voided in 24 hours, 2.5 grams of potassium may be lost very quickly. It thus becomes necessary to replace potassium rapidly and cautiously. Oral feedings of broth, especially bouillon, and fruit juices are very effective in increasing the blood levels of potassium. Potassium may also be given as Ringer's solution or as oral or intravenous potassium chloride solution. Hypokalemia may mask the symptoms of hypocalcemia which may also be present during diuresis. Tetany may be a serious complication of this syndrome. It therefore becomes necessary to replace calcium as well as potassium during the final phase of this syndrome.

It would be logical to expect that with the onset of diuresis there would be a lowering of the blood urea nitrogen, nonprotein nitrogen, and creatinine with a gradual disappearance of uremic symptoms. This is not true in all cases. In many cases of lower nephron nephrosis the nitrogenous metabolites continue to rise as diuresis begins, and the patient who has not exhibited many symptoms of uremia will do so. This situation is a transitory one brought about by the mobilization of large amounts of interstitial fluids in which have been stored massive amounts of the end products of body metabolism. After a few days this transitory uremia will rapidly subside and finally disappear.

The diuretic urine contains large amounts of protein. It becomes necessary to replace these as fast as possible, not only because of the need of maintaining osmotic equilibrium, but protein is invaluable in reversing a negative nitrogen balance into a positive one. Human serum albumin is the therapy of choice. Anemia may be corrected at this stage safely, and washed cells are still preferred to whole blood. As soon as possible, a full dietary regimen should be instituted which should be particularly high in protein, carbohydrate, and fats. Feedings between meals, consisting of eggnog or Protinal in milk, should be given. It is also advisable to continue vitamin therapy and antibiotic therapy well into the convalescent period.

SUMMARY

A discussion of the etiology, pathogenesis, diagnosis, and prognosis of lower nephron nephrosis has been presented.

Treatment during each of the three phases of this syndrome has been discussed. Electrolytic and fluid balance has been emphasized.

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Examination of the Heart*

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The past decade has seen a renaissance of interest in the more accurate and early diagnosis of heart disease. For several decades there was a relative deemphasis on physical diagnosis, owing to increasing dependence on chest x-rays, fluoroscopy, electrocardiography, and others methods of diagnosis. There was a tendency to place abnormal emphasis on diagnosis with instruments at the expense of the art of physical diagnosis. Renewed interest in the art of physical diagnosis, and especially auscultation, has paralleled the advances in cardiac surgery and the advent of the antibiotic drugs. Now cure of heart disease and rather specific preventive methods in heart disease have become a reality.

In the not-too-distant past, it made little difference whether or not a diagnosis was made of coarctation of the aorta, patent ductus arteriosus, pulmonic stenosis, tetralogy of Fallot and several other congenital lesions, or certain rheumatic valvular lesions since there was no cure. Since corrective surgical technics have been devised, there has been concerted effort to make a specific diagnosis of congenital and rheumatic heart lesions. Auscultation is a major factor in this effort—often the deciding finding.

The prevention of subacute bacterial endocarditis with penicillin given before, during, and after all major and minor surgical procedures, including obstetrics, can only be carried out when an organic murmur is detected, indicating the possibility of a superimposed infection on the chronically diseased valve or defect. Prevention of recurrent bouts of rheumatic fever in the patient with a rheumatic valvular lesion is possible only when a diagnosis of rheumatic valvulitis is made on auscultation. The physical findings are necessary and valuable not only in diagnosis but also in prognosis. The knowledge gained from comparing findings at repeated examinations is necessary in therapy in addition to being a guide in the course of the disease and in the prognosis.

Before the physician makes the physical examination of the patient, he often has a preformed idea about the presence or absence of heart disease. This preformed, tentative opinion develops as the history is elicited and as the physician expertly appraises the personality pattern of the patient and the history.

Many times the history is more important in diagnosis than the physical findings, and, indeed, in typical angina pectoris the diagnosis is made from the description of pain and confirmed with the electrocardiogram, the physical examination often being entirely negative. This is especially so in the absence of hypertension. This preformed idea is, of course, a tentative diagnostic opinion soon supported or negated by the physical findings. The physician skilled in physical diagnosis always has specific diagnostic technics for evaluating pathologic changes in certain tissues. There is no substitute for this skill—only mechanical methods which add to the knowledge gained by physical examination.

The time required for conducting the physical examination of the cardiac patient varies, as does the time for history taking. I have spent three-quarters of an hour to an hour on the history of a patient with cardiac neurosis and 15 minutes or less on the physical examination, while at other times I have needed only 20 minutes to take an adequate history but have taken longer for the examination.

Skill in physical diagnosis is based on a thorough knowledge of anatomy, pathology, the physics of sound, and the art of eliciting and interpreting normal and abnormal physical findings through inspection, palpation, percussion, and auscultation. When the physician hears a crepitant or subcrepitant rale, he visualizes moisture or sticky fluid on the alveolar walls with strands of mucus or watery fluid producing a sound like the crackling of dry hair as the alveolar walls separate during inspiration. He knows that this moisture is either a watery transudate, as in heart failure, or it is secretory mucus from the alveolar lining epithelium due to inflammation. One can almost differentiate between the two by the more marked "wetness" of the rales in heart failure. It is such basic knowledge plus skill in eliciting and interpreting the findings that makes the expert in the art of physical diagnosis.

INSPECTION

Frequently inspection is unrevealing, for the patient with heart disease may appear normal in color. In myocardial insufficiency, pallor, duskiness, or even actual cyanosis is frequently present. The pallor of left ventricular failure may arouse suspicion of severe anemia as the cause. More often than not, however, the blood count is normal. Patients in heart failure often show distention of the neck veins. Abnormal carotid and suprasternal pulsations are frequently seen in the chronic hypertensive and in the patient with marked aortic sclerosis and tortuosity. Visible peripheral pulsations of unusual force are noted in the superficial arteries of patients with a high pulse pressure as in aortic regurgitation.

An important sign to look for in taking the history and examining the patient is sighing dyspnea. When sighing becomes a habit, it can produce symptoms and is then known as the hyperventilation syndrome. Any patient noted to sigh fairly frequently should be suspected of this syndrome and questioned further about its frequency and the occurrence of the rather typical symptoms produced by hyperventilation. The symptoms are dizziness, numbness of the extremities, weakness, apprehension, sweating, coldness, precordial pain, and, at times, muscle cramps.

Many times a diagnosis of heart disease, both organic and functional, has been mistakenly made in patients who had the hyperventilation syndrome alone. In order to detect this disorder, the physician must be "hyperventilation minded" just as he must be "hypothyroid minded" if he is to detect hypothyroidism. In general, too, patients who complain of dizziness when they breathe deeply during auscultation of the lungs are candidates for the hyperventilation syndrome since they show a low threshold for registration of vasovagal reflexes.

*Presented at the Fifty-Eighth Annual Convention of the American Osteopathic Association, Toronto, Canada, July 15, 1954.

PALPATION

The art of palpation has been neglected except in the examination of the radial pulse. All valve areas should be palpated to elicit thrills when murmurs are present. The finding of precordial tenderness or hyperesthesia is often very helpful in confirming the suspicion that the heart pain is functional and related to tension states and chronic anxiety. This finding is not to be confused with localized costochondral tenderness so often due to previous trauma or to rheumatic costochondritis. Hyperesthesia frequently is present over most of the precordial area. The degree of tenderness and protest by the patient are often an indication of the severity of the anxiety or tension state present.

BLOOD PRESSURE

The determination of blood pressure is a two-edged sword—a valuable diagnostic aid and, at the same time, a procedure which worsens symptoms or even produces neuroses when improperly used. Both hypertension and hypotension are frequently overdiagnosed. Despite impressive statistics to the contrary by prominent insurance companies and medical clinics, hypotension continues to be overdiagnosed. Unless hypotension follows heart failure, coronary thrombosis, or a cardiovascular accident, the usually diagnosed hypotension is nonexistent. Patients over 40 who regularly have a systolic blood pressure of 100 or 110 should not routinely be labeled as hypotensive. Statistics reveal that in otherwise healthy individuals the outlook for longevity is as good or better in those with a habitual 100 to 110 systolic pressure as it is in those with a so-called normal of about 100 plus age.

There continues to be a universal habit of blaming almost any general symptom on a systolic blood pressure of 100 to 110. This blood pressure level is often a family trait, and in the otherwise normal individual is perfectly compatible with normal circulation and health. The average systolic reading of the oriental is 100, and in him coronary disease and hypertension have an extremely low incidence.

However, hypotension of considerable degree in a patient who has previously been hypertensive may carry a guarded prognosis, depending, of course, on other circulatory findings. The hypotension that accompanies shock in some acute myocardial infarctions is a very serious condition requiring immediate treatment with adrenalin-like drugs.

Insurance companies frequently use 140/90 as top normal levels. The physician can accept higher levels in patients over the age of 50 and, in general, 100 plus age as the top normal systolic level and 100 or less as the diastolic level is a fairly good guide. The fact that does not seem to be apparent to many physicians is that two patients, both aged 60, may have considerable variation in their blood pressure (both below top normal) and yet the one with the lower reading should not be classed as hypotensive. One has a blood pressure of 160/96 and this has been his top normal, having shown a gradual increase with age. The other has a blood pressure of 116/80, this having been near his average pressure ever since the age of 20. Both blood pressures are normal readings. The second patient does not have hypotension nor should he be treated for hypotension. However, if the first patient, whose blood pressure is 160/96, should have an acute coronary occlusion, his blood pressure may drop to average levels of 100/70 when he recovers. This would then be classed as hypotension for him, and certain symptoms might be attributed to this change in tension in the vasculature.

Unfortunately, hypertension is also overdiagnosed in that too much emphasis is placed on one reading or the readings taken at one sitting. Emotion is such a major factor in many patients in elevating the systolic pressure that it must be evaluated before average readings are considered reliable.

The finding of one abnormal blood pressure reading should be followed by unobtrusive rechecks at intervals without exciting the patient. Only after repeatedly finding hypertensive levels should hypertension become part of the diagnosis. Even then, recording of blood pressure levels at every visit should be avoided except in a small percentage of patients. It has been recommended that patients who have uncomplicated hypertension have their blood pressure checked only twice yearly. The intent is to avoid emphasis on hypertension.

PULSE PRESSURE

Pulse pressure is an important finding that sometimes gives a clue to diagnosis. The average normal pulse pressure should be between 30 and 60. Levels below and above this, however, are compatible with good health. A low pulse pressure often results in fatigue and, in certain individuals, predisposes to vascular thrombosis. Often the low pulse pressure is due to a postural drop, present only with the patient in the erect position, due to mesenteric pooling of blood with peripheral vascular deficit. This is corrected by applying an abdominal support or binding to prevent the mesenteric pooling of blood.

Chronic postural hypotension favors the development of coronary disease in susceptible patients. In general, the smaller the pulse pressure, the greater the likelihood of deposition of sludge material on the lining of blood vessels. Pulse pressures that are chronically below 20 are a definite threat to normal tissue physiology and structure.

High pulse pressure should be evaluated as an aid in diagnosis and prognosis. A high pulse pressure with an abnormally low diastolic pressure, as with a blood pressure reading of 180/20, 200/40, 140/30, or 150/0, is almost pathognomonic of aortic insufficiency. The highest blood pressure ever recorded was 400/0 in a patient with aortic insufficiency.

Thyrotoxicosis frequently results in a high pulse pressure but never at the expense of an abnormally low diastolic pressure. Such blood pressures read 160/70, 150/64, 170/72, et cetera. Seldom does the diastolic pressure go below 60 in thyrotoxicosis.

Then there is the large pulse pressure in hypertensives, mostly women, in whom the diastolic pressure is normal and the systolic over 200. Blood pressure readings of 220/80, 200/70, and 240/80 are common (pulse pressures of 140, 130, and 160). This spread in pulse pressure has been described as being due to hypertension in a patient with a rigid, nonelastic aorta. Also, patients with atrioventricular heart block showing apex rates of 40 or less often develop compensatory systolic hypertension with no diastolic increase. This produces a large pulse pressure. In general, such large pulse pressures increase the dynamics of the vascular tree with resultant increase in wear and tear, especially on the proximal thoracic aorta.

HEART RATE AND RHYTHM

The normal heart rate is between 60 and 100 for all practical purposes. There are normal hearts with

(Continued on page 647)

THE JOURNAL OF THE
AMERICAN OSTEOPATHIC ASSOCIATION

Published monthly by the American Osteopathic Association

Publication Office, 100 S. Kenilworth Ave., Oak Park, Ill.
Editorial Office, 212 E. Ohio St., Chicago 11, Ill.
Address all communications to the Chicago Office

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Subscription . . . . . Ten dollars a year in advance

Vol. 54 June, 1955 No. 10

PHYSICAL MEDICINE AND REHABILITATION IN OSTEOPATHY

I. PROPONENTS AND OPPONENTS

The newest specialty group within osteopathic medicine has been granted an affiliate status with the American Osteopathic Association by its Board of Trustees, and since July, 1954, it has been known as the American Osteopathic Academy of Physical Medicine and Rehabilitation. This recognition was also extended to authorize the formation of an examining and certifying agency, the American Osteopathic Board of Physical Medicine and Rehabilitation. These bodies conform to rules and regulations prescribed for such groups by the A.O.A. Board of Trustees. At its December, 1954, meeting, the Board advised the officers of the Academy of P.M. and R., that its name should be changed to that of a college, that its constitution and bylaws should provide for candidate members and associate members, and that its annual meeting should be held in conjunction with the annual convention of the American Osteopathic Association. It is presumed that these procedural requirements will be met and the name of the Osteopathic Academy of P.M. and R. will be changed to the American Osteopathic College of Physical Medicine and Rehabilitation and will be approved at the July, 1955, meeting of the A.O.A. Board.

It is important that very many osteopathic physicians—general practitioners and specialists alike—appreciate that this newest organization can promote the growth of osteopathy generally to a remarkable degree.

The 1955 A.O.A. Convention program has several papers scheduled which deal specifically with physical medicine and rehabilitation as a specialty of osteopathic medicine (see May issue of THE JOURNAL). Convention attendants should plan especially to hear a paper by Dr. W. W. W. Pritchard, Coordinator of Osteopathic Training, College of Osteopathic Physicians and Surgeons (Los Angeles), entitled "The Role of the Specialist in Physical Medicine and Rehabilitation in Osteopathic Medicine Today." His paper will cover the subject fully. It is mandatory for all doctors who question the place of physical medicine in osteopathy to hear Dr. Pritchard. Every osteopathic physician should also plan to see the Physical Medicine and Rehabilitation Center in operation at COPS as well as arrange to visit the P.M. and R. department of the Los Angeles County Osteopathic Hospital. This newest activity of osteopathic medicine needs to be viewed at other than the armchair level.

The Academy of P.M. and R. will hold its annual meeting following the Convention and present a program on July 23. Their program and that presented at the A.O.A. Convention will afford a dual opportunity for these specialists to state their case, outline their plan, and point out its peculiar significance to osteopathic medicine. They will have a chance to answer questions put by those who are troubled by physical medicine and rehabilitation as a specialty of osteopathic medicine, but who are willing to suspend judgment until they objectively re-examine its claims.

Criticism of this newest specialty of osteopathic medicine is not directed at its leaders who are osteopathic physicians of high repute, and who acknowledgedly possess technics and skills that differ from those commonly associated with osteopathic manipulative treatment. Personal elements are not involved in these conflicts which stem from varied attitudes and convictions.

There is a small sector within the profession which sees in physical medicine and rehabilitation a subtle attempt to confine the practice of osteopathic medicine to the technics of manipulation and physiotherapy—a modification of the effort to equate osteopathy with manipulative technics, an approach as old as the profession itself. At the opposite pole are occasional critics who would have none of physical medicine because they view the specialty as an equally subtle method of doing away with osteopathy altogether by containing it within physical medicine.

Other critics of this specialty program belong in neither of these contradictory categories. Some are uninterested in physical medicine and rehabilitation, and dismiss it as of little moment. Physical medicine, they say, is a fancy name for physiotherapy—modalities by which heat is applied, and "heat is heat"—and rehabilitation, well, that is largely exercise.

Some attack the A.O.A. because physical medicine has been granted specialty status. They see the specialty as a menace, contradictory in every aspect, and confusing to young doctors of osteopathy. These particular critics are typified by one able osteopathic physician who wrote, admittedly facetiously, in a personal communication to the Editor that "we need physical medicine [in the osteopathic profession] as we need asafetida." He immediately countered with the offer to produce "technical argument" against what he termed

to be "a mad rush to overspecialize" upon the part of the osteopathic profession and its national organization.

Critics of the osteopathic physical medicine and rehabilitation program are alike in that they imply that as a specialty it has no historical roots, and no true relationship to organized medicine, much less to organized osteopathy. They rate physical medicine and rehabilitation as an "answer" to osteopathy. These critics dismiss the old-school specialty of physical medicine as a rationale for the expenditure of more than a million dollars in its decade-long exploration of structural medicine, a study largely instigated through the interests and efforts of Bernard Baruch. Mr. Baruch's real purposes are seen as defeated. Thus, physical medicine is dismissed as born of subterfuge and cradled in evasion. These same critics see doctors of osteopathy who are P.M. and R. specialists as merely trying "to cover up for their profession's deficiencies, now that it has so largely forsworn the osteopathic approach."

Such are the positions of many of the critics of the program of physical medicine within osteopathic medicine. The lines between these minority sectors (for osteopathic physicians in physical medicine and rehabilitation are also a minority) are sharply drawn. Opponents and proponents—and we hope to draw in many who for many reasons are disinterested—need to precede any discussion of their respective points of view with a broadly set up semantic introduction to the entire project. Although these groups speak the same language, it is most often with little understanding of its meaning as used by the one or the other. Such an introduction put in question form should afford a body of definitions as a point of departure for any genuine examination of the issues involved.

II. QUESTIONS AND ANSWERS

The questions below have been asked directly or have been implied by many osteopathic physicians whose opinions were sought about the place of physical medicine within the profession. The answers likewise stem from many sources. They are not dogmatic in intent, but are set down with the hope that they will lead to a full interplay of opinion within the profession.

1. *What is physical medicine?*

The newest of the organized specialties, physical medicine is basically one of the oldest methods of diagnosis and treatment. It has its roots in primitive medical concepts and is related to a mechanical approach to disease that goes back hundreds of years. As acceptable as any is this definition of physical medicine authorized by the Veterans Administration:

> . . . the employment of the physical and other effective diagnostic and therapeutic properties of light, heat, cold, water, electricity, massage, manipulation, exercise and mechanical devices for diagnosis, treatment and for research.

Rehabilitation has been defined by the same agency as

> . . . the employment of physical medicine, technics of psychosocial adjustment and vocational retraining for the purpose of aiding the patient to achieve the maximal function and adjustment and of preparing him physically, mentally, socially and vocationally for the fullest possible life compatible with his abilities and disabilities.

As a specialty within osteopathic medicine, physical medicine and rehabilitation has been officially defined as consisting of and including

> . . . the use of all medical measures, especially those in the physical field, which will hasten the recovery and restoration of the ill or handicapped to the fullest physical, mental, social, vocational, and economic usefulness of which he or she is capable.

2. *What is the difference between physical medicine and what formerly was termed physiotherapy in old-school medicine?*

Physiotherapy, like the term "drug therapy," merely indicated the type of agent employed, that is, a physical modality. Physical medicine (like pharmacology) indicates an over-all concept and a continuity of approach to the selective application of methods and modalities. It is the precise usage of every device and modality of proved value in diagnosis and treatment. Physical medicine and pharmacology suggest conceptual thinking; physiotherapy and drug therapy are merely terms.

3. *How can physical medicine (old-school) logically include "manipulation" as one of its modalities?*

Manipulation, particularly manipulation of joints, is nothing new in medicine. It was a part of the technics of primitive medicine. The bonesetters of Britain and the continent long antedated Still. Manipulation historically is part of medicine, hence it is properly included within physical medicine, whether as an old-school specialty or one of osteopathic medicine. This statement does not imply that traditional bonesetting or manipulation within the physical medicine practiced today can be classified as osteopathic manipulation. The converse of this statement, however, is true, for manipulation so used is a generic term while *osteopathic manipulation* is a specific term. "Bonesetting" and "adjustment" as used by various nonmedical groups, manipulation as applied to the present usages of physical medicine, and osteopathic manipulation, all come under the generic classification of manipulation. To confuse a generic usage of the term with its specific usage, would be as absurd as to equate the administration of digitalis by Withering (1741-1799) with its exhibition today by the internist.

4. *Will the classification of osteopathic manipulation specifically under manipulation generally tend to destroy the distinctive relation it bears to those osteopathic principles (osteopathic concept) which form the basis of osteopathy as a system of medicine?*

On the contrary, such a classification will reveal its distinctiveness. Osteopathy as a system of medicine emphasizes the importance of the musculoskeletal system as the primary machinery through which man expresses his life. The reciprocal interaction of the musculoskeletal system with all other systems, the effect of musculoskeletal lesions as harmful agents or noxious influences in promoting disease—all these factors combine to make osteopathic manipulative treatment a distinctive and unifying agent in the maintenance of health

and the treatment of disease. So considered it is an agent distinctly different in its origin and type from manipulation as currently employed in physical medicine. The emphasis upon the difference is not for the sake of difference, but because musculoskeletal lesions have etiologic, diagnostic, and therapeutic implications which have resulted in the development of osteopathic manipulative procedures and have served both as a rationale for and a guide to their use.

Osteopathic manipulation is an integral part of physical medicine as a specialty of the osteopathic school of practice, and it is legitimately classified and its technics taught as a part of that specialty. Incidentally the premise can be well supported that osteopathic manipulative technics should always be classified for teaching purposes under a department of physical medicine and rehabilitation where an osteopathic college has such a department. The teaching of osteopathic principles or osteopathic philosophy, however, should always fall into the department where it belongs historically—general medicine.

5. *Will the establishment of a specialty college of physical medicine and rehabilitation tend to place osteopathy within physical medicine?*

It would, if those doctors of osteopathy who equate it (actually or in effect) with its distinctive technics of treatment were correct in their assumption. If "osteopathy" meant osteopathic manipulative treatment, then osteopathic physical medicine logically would become inclusive of *osteopathic* manipulation, hence osteopathy. Whereupon the tail would wag the dog!

6. *Specialists in osteopathic physical medicine and rehabilitation stress the distinctiveness of the osteopathic approach and the superior effect of osteopathic manipulation—might that emphasis lead to its total transplantation into the specialty field and its abandonment as an integral part of osteopathic practice (osteopathic medicine would go on without either its distinctive philosophy or its therapeutic technics)?*

In variant form (basically, as the premise implied in number 5 above) for more than a half-century, this question has appeared and reappeared, always from the same ground of assumption: equating osteopathy with osteopathic manipulative technics. Physical medicine cannot contain osteopathic medicine; axiomatically the part cannot contain the whole. As an osteopathic specialty, physical medicine will base its entire rationale upon osteopathic philosophy, thus possessing the inherent right to utilize all distinctive osteopathic technics of treatment. To presume that this usage will leave osteopathy void of the foundation of its existence is to fail to conceive the depth of the osteopathic movement within medical history, past, present, and future. If the presumption were true, osteopathy would indeed be a thing of little moment, cut off from the continuum of medicine even before its inception, and destined to an end, not only as an organized sector of medicine, but philosophically, scientifically, and technologically.

Osteopathic physical medicine, however, through its employment of manipulative technics that are distinctly osteopathic will result in an enrichment of itself far beyond its possibilities within old-school medicine, for it will open up opportunities now lacking for the further development of those technics. But greater, the measured technics of physical medicine developed within the last decade will prove an effective cross-fertilization that will result in an enrichment of osteopathic medicine generally.

Specialization in medicine, for all of its divisiveness and the problems that arise therefrom, was inevitable. Its development within osteopathic medicine is an equally destined thing. But osteopathic specialization in the physical medicine and rehabilitation field is freighted with less problems, fewer contradictions, and is much more a natural and evolutionary development than is true of any other specialty within osteopathic medicine, except, perhaps, that of osteopathic surgery.

7. *Is there a possible conflict of fields of practice or of interests between the physical medicine and rehabilitation specialists and those doctors of osteopathy who, while recognizing osteopathic medicine as the most nearly complete system of medicine, choose to confine their therapy to osteopathic manipulative procedures and become highly skilled therein?*

There is no conceivable conflict between this specialty group and those individuals who by aptitude develop very superior manipulative skills—inheritors of the profession's richest tradition. Many of these doctors are members of the Academy of Applied Osteopathy. Without doubt the growth of physical medicine and rehabilitation as a specialty within osteopathy will lead to the wider development of distinctively osteopathic technics of manipulation to be applied generally. As a matter of fact, the Academy of Applied Osteopathy and the Academy of P.M. and R. could work closely together in the further evolution of osteopathic manipulative technics.

8. *If osteopathic principles and technics have so important a place in osteopathic physical medicine, is there any real place or need for the specialty per se in this profession?*

The answer is that there is a definite place for physical medicine and rehabilitation in any unlimited system of medicine, osteopathic as well as old school.

The great weakness of physical medicine is that it tends to be "gadget medicine." It has been correctly observed that the concept of specificity in disease causation and in therapy absolutely dominates modern medicine—so that a few medical writers of authority are daring to call it "etiological" medicine. It is inevitable that old-school physical medicine should be largely etiologic and curative and characteristically without a unity of approach to the problems it seeks to solve.

Specialization, however, is demanded in physical medicine becauses many of its modalities are precision instruments, requiring exact dosage. But that is not enough. The specialty to develop its potentialities—richer than any other special field of medicine except psychiatry—requires much more. It must go far beyond its present etiologic approach and curative intent. Osteopathic principles and technics applied to physical medicine and rehabilitation as a specialty could result in breaking down the divisibility and segmentation that

characterizes all specialty medicine. Such an application might well point the way for these principles and technics to become a more potent factor in other specialty fields where such an influence is so sorely needed.

Osteopathy cannot become osteopathic physical medicine and rehabilitation unless the profession itself fails to place in the central position the real issue—not that of recovering a golden age of an osteopathic past or looking only to a medical Utopia of tomorrow—the necessity of working toward a solution of the problems of osteopathic medicine today as they arise within the total structure of medicine, itself problem-beset.

III. FRESH INSIGHT AND NEW GOALS

It has been suggested recently that the primary weakness of the osteopathic movement today is its self-satisfaction with its past insights and its established goals. The questions that have arisen concerning physical medicine and rehabilitation are only additional evidence that the time has come when the profession is called upon to re-examine itself historically and reassay its purpose. To do this osteopathic medicine must be seen *from the outside as well as from the inside.*

Osteopathy has long passed its "adjunctive" days, a highly artificial, totally unrealistic period when all agents, modalities, and methods of treatment were seen as "adjuncts" to osteopathic manipulative care. That period was one with the time when many osteopathic physicians considered osteopathy to be a self-contained entity unrelated to medicine generally and set apart from its development. Such was a popular inside view.

Osteopathic philosophy (the "principles" of osteopathy or the osteopathic concept, as you will) was so overshadowed by its distinctive therapy (osteopathic manipulation) that the idea of an "adjunctive" therapy appeared logical. That premise granted, osteopathy became a mutation, a sport off the centuries-old vine of medicine. Such was the thinking within a large sector of the profession—a view that became acceptable from the *inside.* That view was encouraged from the *outside* and was made a matter of political maneuvering, a clever strategy by which the osteopathic profession was forced into a position that was as untenable as it was historically untrue. So successful was the strategy that a broad program of education inter-intra-extra-professionally has become a continuing necessity if the osteopathic profession is to fit itself to fill the place that is being so rapidly created for it. From the outside, it is most important that the osteopathic movement be seen in its full historical significance as a movement within medicine, influencing it and being influenced by it.

The converse is true. No view of osteopathy can be a total view unless it is seen also from the inside. For that reason it is almost impossible for a doctor of medicine to comprehend osteopathy. It is the inside and outside views which create a synthesis that makes for truthful and logical answers to questions which press upon the profession for answers.

These questions that concern physical medicine and rehabilitation are but archetypes—if we find the answers to this particular problem, we shall find ourselves prepared to solve many other issues that are more complex and demanding.

The time has come for the proponents of physical medicine and rehabilitation within osteopathy to explore all the methods of communication by which they can inform and educate osteopathic physicians concerning the startling relevancy of their program to this profession. The program has already demonstrated its worth in widening the appreciation of many osteopathic students to a structural approach to health problems as well as to the effectiveness of osteopathic manipulative therapy.

By far the greater number of doctors of osteopathy are, however, unacquainted with the tremendous development within the last decade of the technics of physical medicine and rehabilitation—this same lack of information is found among doctors of medicine who are largely noncritical. Too many doctors know no facts about which either to be critical or enthusiastic.

Progress in physical medicine and rehabilitation in osteopathic medicine will be found to move slowly and deliberately. Just now it is compelled to wait on a growing knowledge of the extraordinary place which the specialty could occupy and the revitalization it could render to a profession that is already showing a dangerous self-satisfaction. It is ironical that this particular specialty, the one most natural to our profession, is the one to be the least graciously received.

As is common in all happenings in every field of man's activity, we become so taken up with many things that we miss, or misconstrue, the meaning and significance of the happening itself. This broad introduction to one happening in organized osteopathy (the rise of physical medicine and rehabilitation) is intended to serve as a semantic basis from which to explore its meaning. The presentation could serve as a method by which the truth can be sought by those with varied points of view about many problems that harass organized osteopathy. And truth should always be sought dialectically, which means to this writer, to permit the free expression of "yes" and "no," until a "yes" has been reached which is hardened in the fires of many "noes." The end result is the union of all of the elements of truth brought out in the dialectic process (the discussion).

This profession needs most of all to put aside its fears on the one hand and its self-satisfactions on the other and recapture the spark of divine discontent that led to its emergence from the medical practice of the turn of the century to become a movement for the reorientation of medicine. That was and is its ultimate purpose, and that is its destiny. Osteopathy needs reinterpretation and rephrasing within the meaning and language of the mid-century, but not as a thing apart from medicine. Physical medicine and rehabilitation—organized within and without the profession—represents but one phase of osteopathy's continuing challenges to the fulfillment of its purpose.

SUDDEN DEATH DURING INFANCY

Several thousand young infants in apparently good health die suddenly and unexpectedly in the United States every year, according to authoritative estimates. These deaths have been attributed to accidental suffocation, enlargement of the thymus, adrenal hormone imbalance, allergic reactions, various forms of infection such as interstitial pneumonia and focal bronchopneumonia, and, recently, low gamma globulin levels. Autopsy reports have, in the majority of cases, revealed little of significance other than respiratory pathology present in greater or lesser degree. The most careful investigation of the surroundings and the circumstances and detailed gross and histologic postmortem studies have failed to explain adequately either the suddenness of the deaths or the absence of significant symptoms. And no scientific study can in any way diminish the grief, the shock, or the feelings of guilt of parents of babies who die in this unexplained and even mystifying manner.

An important article, "Suffocation or Sudden Death of the Infant," by F. J. Thompson is published on pages 607-612 of this issue of THE JOURNAL. It reviews the literature on the various theories of causation, especially allergy and agammaglobulinemia, and discusses a possible means of prevention, the routine administration of gamma globulin to selected infants during the first months of life. As Dr. Thompson points out, the only approach to the solution of this problem is the preventive or prophylactic approach. He concludes:

> The various questions raised in this paper present some of the facets of the problem which have to be resolved. Many investigators are vitally interested in this subject and some are working alone, some thinking alike, and others thinking along different channels. Only through concerted and coordinated efforts, particularly in the large centers with concentrated pediatric services, can progress be made more rapidly in determining the underlying causes of these sudden unexpected deaths in infants. Only when such efforts have been made will we have a more positive approach to the prevention of these deaths.

Notes and Comments

Salk Poliomyelitis Vaccine.—

The action of the United States Public Health Service halting the inoculation program with Salk vaccine raised many more questions than can be answered or that probably will be answered publicly. It is important, however, that physicians inform themselves on the question of "live" *vs.* "dead" vaccines. The issue will not be determined by this country's medical opinion, which on the whole is against live polio vaccine.

Albert B. Sabin, M.D., College of Medicine, University of Cincinnati, considered as one of the ablest investigators of polio in the United States, is not discouraged by opposition to the use of live vaccine. For years he has been experimenting with the three known types of polio virus. He has isolated them and modified them so as to produce mutants which breed true. He reports giving his mutants to twenty-six volunteers in a federal reformatory between the ages of 21 and 30, a highly susceptible period of life. The volunteers were mildly infected with polio to the degree that millions of the population have been mildly infected but never knew it.

The Sabin vaccine had been previously given to chimpanzees which were not infected regardless of the route of administration. But as long as the living mutants can produce any discernible evidence of polio in the human, no one would suggest its use, and certainly Dr. Sabin does not do so. Before a living virus is used an answer must be given to the question: If a virus mutated once, even in a humanly favorable direction, may it not mutate again in one that is lethal? Yellow fever vaccine is an example of the highly successful use of virus mutants; the same stability must be assured for the polio mutants, and it is to that end that Dr. Sabin is pursuing his experimentation.

A live vaccine immunity of at least 40 years is claimed—that claim, too, is far from proof. But, granting a measured success to the Salk vaccine, as was pointed out last month in THE JOURNAL, is revaccination every 2 or 3 years of a tremendous body of people a practical thing?

To the United States Public Health Service, through its Division of Biologics Control of the Nashould go for decisive action in a situation in which tional Institutes of Health, the highest commendation there appeared to be complicating factors operating that were quite beyond their control.

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Our Health Future.—

"Notes and Comments," page 533 in the May JOURNAL, took notice of the Thirty-Fifth Annual National Health Forum recently held in New York City, at which a noteworthy address was delivered by Dr. C. J. Van Slyke, Associate Director, National Institutes of Health, stressing the study of "the whole man in relation to other men and to the environment in which they live."

Among other important addresses was one by John C. Bugher, M.D., director of the Division of Biology and Medicine, U.S. Atomic Energy Commission, in which he said that although a "tremendous revolution" has already occurred, we merely stand on the threshold of a new and exciting era of advances in biochemistry. The study of dynamic characteristics of intracellular processes within the living and unharmed cells instead of the morphologic approach, an attack on the physiology and pathology of aging and on the degenerative and neoplastic diseases, increasing productivity of food

crops, and speeding up genetic improvement of plants are developments to be expected.

At the final session representatives of the more than forty-five national organizations concerned with improving the health of the people of the nation were warned by Howard A. Rusk, M.D., that "A civilian disaster of the magnitude possible today could put an incredible load on the available physicians." He pointed to the National Health Council as an organization which more than anything else ". . . is to demonstrate how democracy works." He referred to the health professions as ". . . demonstrating to the world the essence of democracy," and said further, "We have a great opportunity and a great responsibility, for we have a common language that, regardless of race or creed or color or politics or bamboo curtains, can still be spoken."

Our readers will recall that Dr. Rusk is exercising the kind of medical leadership that makes him the spokesman for the medicine of tomorrow—a comprehensive medicine. He is an associate editor (medical) of the *New York Times;* a chairman of the Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, and chairman of the Health Resources Advisory Committee, Office of Defense Mobilization.

The new president of the National Health Council is Hugh R. Leavell, M.D., professor and head of the Department of Public Health Practice, Harvard School of Public Health. Philip E. Ryan continues as the Council's able executive director. Much of the broad program and splendid accomplishments of NHC are made possible through its sustaining members, the Equitable Life Assurance Society of the United States and the Metropolitan Life Insurance Company.

Appointed as delegates from the American Osteopathic Association to attend the NHC and its public meeting, the Forum, were Drs. Roswell P. Bates, Alexander Levitt, and Floyd F. Peckham.

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Second Thoughts.—

The May, 1955, issue of the *Scientific American* carried a semihistorical-scientific article by René J. Dubos entitled "Second Thoughts on the Germ Theory." It is a continuation—in more popular vein—of the ideas expressed in the article, "The Gold-Headed Cane in the Laboratory," published in *Public Health Reports,* April, 1954, and reprinted in the July, 1954, issue of THE JOURNAL. Many readers will recall that a short item dealing with Dr. Dubos' philosophic approach to medicine appeared in *Time* for November 1, 1954. The *Time* story was widely commented upon by osteopathic physicians in letters to the A.O.A. Editor. René Dubos is a member of the Rockefeller Institute for Medical Research in New York City; in 1939 he isolated an antibiotic substance now called tyrothricin.

In his *Scientific American* article the author emphasizes the heated controversy that preceded the general acceptance of the germ theory of disease in the 1870's, physicians especially being among the die-hards who were critical of Pasteur and Koch. Once the microbe was acknowledged as "a constant and ubiquitous component of the environment" of man and animals it became evident that a population which survives and flourishes does so because "its members are endowed with a high degree of natural resistance to the microbes normally present in that environment." The classical doctrines of immunity resulted in no understanding of "the agencies responsible for natural resistance to infection and of the factors that interfere with the operation of these agencies."

Dr. Dubos re-emphasizes the importance of "a new look at the biological formulation of the germ theory," now that the property of virulence of germs in relation to a population is coming to be thought of as *ecological,* which, simply stated, is man's ability to adapt to his peculiar total environment which includes his fellowman, or, as Dubos puts it, the question of "Whether man lives in equilibrium with microbes or becomes their victim depends upon the circumstances under which he encounters them."

Citing the observations of James Reyniers and his colleagues at the University of Notre Dame—animals born and raised in a sterile environment died when they were exposed to common bacteria such as are always present in a normal environment—Dr. Dubos suggests that "the mechanisms responsible for natural resistance are in general most effective under the narrow range of conditions constituting the 'normal' environment in which the population has evolved." He suggests that among the factors that may upset the equilibrium are irradiation, metabolic abnormalities, treatment with antimicrobial drugs, and psychosocial factors. The article ends with a quotation from John Caius, English physician (1510-1573), writing on the "sweating sickness" which was rampant in England in 1552: "Our bodies cannot . . . be hurt by corrupt and infective causes, except ther be in them certein mater apt . . . to receive it, els if one were sick, al shuld be sick."

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Research Arm.—

The principal research arm of the U. S. Public Health Service is the National Institutes of Health with the broad purpose of seeking better methods of preventing, diagnosing, and treating the major killing and crippling diseases of man, such as mental illness, neurologic disorders, cancer, heart disease, and arthritis. A fifty-six-page bulletin, "Medical Research Accomplishments," just published by the Social Legislation Information Service, is devoted to reports on the seven Institutes in 1954.

The program of the National Institutes of Health extends far beyond the research conducted in its own research laboratories. Approximately two thirds of approximately $80 millions appropriated annually to NIH support research and training in nonfederal institutions. Fellowship awards are made to develop research talent, traineeship awards to provide special clinical training, and grants to assist in research and in improved teaching or training in certain medical areas; and all

of these awards are participated in to a greater or lesser degree by the osteopathic profession.

The bulletin reports in concise, not too technical language the highlights of research progress in 1954 by the Institutes of mental health, neurologic diseases and blindness, cancer, heart diseases, arthritis and metabolic diseases, microbiology, and dental research.

A resume of the research advancement in microbiology (the field of the National Microbiological Institute) is especially interesting, just as its work in germ-free medical research is especially significant. The objective is to raise and make available for experimental use small laboratory animals which have been freed of the bacteria harbored by conventional or contaminated animals. Such animals and their progeny are maintained through successive generations in sterile surroundings, this includes their living quarters, the air they breathe, and the food they eat. In 1952 this Institute initiated a germ-free study in cooperation with University of Notre Dame's laboratories in biology, the project referred to in the Dubos article commented on in another paragraph of these Notes and Comments.

Eventually, these studies might be one of the doors which will open into a fundamental investigation of the disease process, apart from disease as it is now conceived as purely a problem of etiologic medicine. There seem to be relational possibilities here to developing osteopathic concepts as seen by Korr and his associates, now at work on fundamental osteopathic research.

Osteopathic teachers, writers, and physicians might well inform themselves on social advance in fundamental medicine research by securing this copy of the bulletin. Write to the Social Legislation Information Service, Inc., 1346 Connecticut Avenue, N.W., Washington 6, D.C.

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Anatomical Pattern.—

"Anatomical Pattern As the Essential Basis of Sensory Discrimination," the Forty-ninth Robert Boyle Lecture, by W. E. Le Gros Clark, Professor of Human Anatomy, University of Oxford, has been called to the attention of osteopathic physicians by Dr. George Grainger, Tyler, Texas. This little sixteen page pamphlet, published by the Blackwell Scientific Publications, Oxford, was also published simultaneously by Charles C Thomas, 301-327 East Lawrence Avenue, Springfield, Illinois. Doctors interested in an anatomical consideration which goes beyond fact would find such an approach as this stimulating:

". . . the fundamental riddle of biology [is] the riddle of form, a riddle which is involved in the problem of the orientation and organization not only of nervous tissue but of all kinds of living tissue. It concerns not only the construction of organized patterns during development and growth, but also the maintenance of their integrity during the whole of life, in spite of the continual renewal and substitution of their material substance which occur unceasingly in the process of metabolic interchange. It is important to recognize this simple fact.

In one sense there is nothing static about any anatomical unit in the living body. For example, it is well known that even the inorganic constituents of bone are being continually removed and replaced. The material of your thigh-bone to-day is not the same as the material of your thigh-bone to-morrow, and yet, in its contour and architecture, the bone outwardly preserves its identity. . . .

But not only does this process of change affect anatomical units as a whole, it involves the very molecules out of which living matter is constituted. In the phenomenon of transamination, nitrogen is being continually shifted on in living tissue from one organic molecule to another in an everlasting chain of "hand to hand" transport. Thus, not even the ultimate elements of living substance are stable. Yet the pattern to which they conform remains the same. . . .

It is as though, when we look at the living body, we look at its reflection in an ever-running stream of water. The material substratum of the reflection, the water, is continually changing, but the reflection remains apparently static. If this analogy contains an element of truth, and we are justified in regarding the living body as a sort of reflection in a stream of material substance which continually passes through it, we are inevitably faced with a very profound question indeed—what is it, in fact, a reflection *of?*

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"The Human Animal."—

One of our researchers in the physiologic sciences has emphasized the significance to all of our doctors of a book published in 1954: "The Human Animal," by Weston LaBarre, the University of Chicago Press. Calling the book "exciting, informative, and charming," our correspondent rates "The Human Animal" as an especially "valuable volume" for our readers.

Professor LaBarre is a member of the Department of Anthropology at Duke University and, although a young man, has broad academic training and wide experience, including field work on several continents. In his book Dr. LaBarre presents a synthesis of the sciences dealing with man by integrating in a lively but trustworthy fashion human biology, cultural anthropology, psychiatry, and related fields. This he does because it is his firm conviction that "the whole trend of 20th Century Science is plainly toward integration, a fact indicated in the very names of the new disciplines: psychosomatic medicine, biochemistry, psychobiology, and the like."

The author sees the culture of human beings—"and cultures are possessed by human beings alone"—as ultimately resting upon the biologic traits of Homo sapiens, emphasizing that *"Man's 'human nature' derives from the kind of body he has."* Starting in the earlier chapters of the text with man's basic organic prehistory, LaBarre moves forward swiftly through its total of fifteen chapters, selecting and synthesizing to give a proper picture of man. He ends upon the note, "Three minutes to midnight," a word quietly and inspiringly written, thoughtful and thought-provoking. And for those readers who may be disturbed by the thought of a "serious" book, the assurance is that first this is indeed a charming book!

Some will question the importance of this book to themselves: They will counter with a 600-year-old observation that of the making of books there is no end and, further, that this is merely another book being recommended to physicians who do not have time even

for professional reading. Our correspondent has the answer, pointing out that here is a volume which in essence is occupied with the same concepts and approaches which characterize the osteopathic movement: "A deepening understanding of *man* and of *his* life in *his* environment . . . and a concern with the factors associated with human biology and arising from human life which impair or threaten the individual's capacity to live human life, and with the management of these factors."

Order "The Human Animal" from your bookseller or directly from the University of Chicago Press, price $6.00. Its reading is a rich experience.

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CD and CDD.—

The effect of chronic disease in changing the practice of medicine is shown in the following excerpts from an editorial in the January, 1955, issue of *Health News*, a publication of the State of New York. The editorial is signed by Herman E. Hilleboe, M.D., Commissioner, N. Y. State Department of Health.

Since early in the twentieth century, public health workers have concentrated much of their effort on the control of acute communicable diseases. Today, these once deadly diseases no longer present a major threat to the health of babies and children. Widespread and continuing application of public health measures such as improved sanitation, clean food and milk, and immunizations against smallpox, diphtheria, whooping cough, and tetanus has helped to overcome the hazards of these and other communicable diseases. . . .

CD [chronic disease] has been replaced as our principal public health problem by a more formidable adversary, CDD—chronic disease and disability. CDD includes heart disease, cancer, diabetes, arthritis, and many other long-term illnesses and disabling conditions which are difficult to treat and, in most cases even more difficult to cure. . . .

In meeting the problem of CDD in the older age groups, geriatrics is assuming an increasingly important role. Rehabilitation of older persons is commanding more and more of the attention of specialists in physical medicine and their associates in physical therapy, occupational therapy, vocational guidance and training. . . .

By skillfully blending geriatrics with rehabilitation service, we can accomplish lasting benefit in a surprisingly large proportion of older people. Disease and disability are always present among many of our older citizens; the major task is to keep CDD from progressing and to help these people to make physical and psychological adjustments.

Proposed Amendments to the Bylaws of the American Osteopathic Association

R. C. McCAUGHAN, D.O.
Executive Secretary

(The following proposed amendment is published at the request of Dr. Ira C. Rumney, chairman of the Committee on Ethics and Censorship. It would change the method of procedure in cases of charges of violation of the Code of Ethics.)

Article VIII—Duties of Board of Trustees

Amend Section 7 by deleting the entire section and substituting therefor the following:

The Board of Trustees shall have final decision on all questions of ethical or judicial character and shall investigate through the Committee on Ethics and Censorship all matters of an ethical or judicial character and all charges of violation of the Constitution, Bylaws, or Code of Ethics, or of grossly unprofessional conduct of any member. The Committee on Ethics and Censorship shall have the power to cite the member to appear before the Committee to answer such charges, provided that, upon final conviction of any member of an offense amounting to a felony under the law applicable thereto, such member shall automatically be deemed expelled from membership in this Association; conviction shall be deemed final for the purpose hereof when affirmed by an appellate tribunal of final jurisdiction or upon expiration of the period allowed for appeal.

Upon completion of its investigation, the Committee on Ethics and Censorship shall submit to the Board of Trustees of this Association for final action a summary of the investigation together with the Committee's recommendations.

If a member shall have been suspended or expelled from a divisional society because of breach of the Code of Ethics by proper action of such a divisional society, the Committee on Ethics and Censorship shall review such decision and, after careful study and any necessary investigation, submit to the Board of Trustees for final action at the next succeeding session, a summary of its findings and its recommendation. The divisional society shall have the right of appeal to the Board of Trustees from an adverse recommendation by the Committee on Ethics and Censorship.

If the said Board of Trustees shall find a true bill of particulars, such member shall forfeit all privileges of this Association for the further period during which said member shall continue to be suspended or expelled from said divisional society. Such member shall retain the right to present additional evidence of his membership acceptability to the Committee on Ethics and Censorship, for review at subsequent sessions of the Board of Trustees of this Association and, if their findings warrant, his membership in this Association may be reinstated by a three-fourths vote.

(The following proposed amendments are published at the request of Dr. Ira C. Rumney, chairman of the Committee on Ethics and Censorship. They would provide for the establishment of committees not listed in the Bylaws and would permit changing the committees as circumstances warrant without having to amend the Bylaws.)

Article IX—Departments, Bureaus, Committees, and Sections

Amend Section 1, by inserting a new sentence at the end of the first sentence to read as follows:

Committees may be established by the House of Delegates or the Board of Trustees so as to efficiently carry out the work of the various bureaus as outlined by the House of Delegates and/or the Board of Trustees.

Amend Section 2 by placing a period after the word "Welfare" in the first sentence, by deleting the remainder of the sentence, and by inserting a new sentence which will read as follows:

Committees may be established by the House of Delegates or the Board of Trustees so as to efficiently carry out the work of the various bureaus as outlined by the House of Delegates and/or Board of Trustees.

(Continued from page 638)

rates below 60 and above 100, but the majority fall between these two figures. There still exists among the laity the belief that the normal heart should beat 72 times per minute, and many physicians still show concern over heart rates in the nineties. The normal heart is capable of tachycardia for years without showing evidence of strain or failure.

Many patients have been observed who have had uncontrolled hyperthyroidism and sinus tachycardia for years without evidence of cardiac enlargement or of heart failure. When the heart beats more rapidly, the minute stroke volume output decreases with each beat, thus conserving work expenditure. This adaptation permits the heart to beat rapidly for long periods with no unusual effects. Thus the normal heart tolerates paroxysmal tachycardia with rates of 180 to 220 for days and even weeks without failure.

Rapid heart rate is, of course, one of the most common causes of heart consciousness and palpitation. It is also one of the first trigger responses of the heart to emotion. Undue attention to ordinary sinus tachycardia by the physician may be a major factor in producing a cardiac neurosis in the susceptible patient.

Over the past 3 years, I have seen in consultation and on recheck examination three female patients with regular but rapid heart rates between 120 and 140 at all times. All three were unmarried; two were in their fifties and one was 45. All three were hospitalized more than once and completely studied. None was thyrotoxic; none had organic heart disease. Our final diagnosis in all three patients was neurocirculatory asthenia. While under observation, all three received digitalis, quinidine, and Pronestyl in trial courses, and none showed a slowing of the pulse on medication. All three are still free of heart failure and cardiac enlargement. Two still work, the third has retired but takes care of a two-story house. The latter patient seldom has a heart rate under 130.

Heart rates between 90 and 110 are often noted, especially in women at the menopausal age, with other evidences of sympatheticotonia such as hot flushes. This finding should be de-emphasized by the physician or not mentioned at all when all other cardiac findings are normal.

Heart rates in the fifties in otherwise normal hearts are usually an evidence of vagotonia, athletic training, or hypothyroidism. Organic atrioventricular heart block eventually results in bradycardia, usually the rates being below 50. One such patient had an apex rate of 18 for several years before he expired, and rates between 30 and 50 are common in this disorder.

AURICULAR FIBRILLATION

This arrhythmia is not difficult to diagnose at the bedside or in the office if one remembers that it is an absolutely irregular heart with a pulse deficit. Seldom is it necessary to resort to the electrocardiogram to confirm the diagnosis. It is most important to diagnose this irregularity since it does respond to digitalis and/or quinidine and, if uncorrected, markedly decreases the efficiency of the heart as a pump, leading eventually to failure and the formation of intra-auricular thrombi, which represent potential emboli. This arrhythmia occurs most commonly in rheumatic heart disease (especially mitral stenosis), coronary heart disease, and thyrotoxicosis.

EXTRASYSTOLIC ARRHYTHMIA

This is one of the most common irregularities and should be recognized easily by the so-called regular irregularity and the same sequence of dropped beats at the radial pulse. Ventricular extrasystoles are easier to recognize than auricular premature beats since they produce a more obvious change in heart sounds. In the normal heart, extrasystoles, even though frequent, do not significantly impair the efficiency of the heart as a pump. When frequent in diseased hearts, they influence cardiac physiology adversely.

Most extrasystolic arrhythmias are of benign origin and are due to tobacco, coffee, allergy, neurogenic factors, and reflex from upper abdominal disorders, especially gallbladder disease. Again, undue emphasis by the physician on such an arrhythmia in a patient without heart disease may initiate a train of symptoms psychosomatic in mechanism but often difficult to dislodge as the pattern becomes ingrained in the patient's psyche. Extrasystoles may occur regularly every second, third, or fourth beat, or at random. When they occur every other beat, the irregularity is referred to as bigeminy or coupling.

This combination, in which every other beat is an extrasystole, occurs in normal hearts as well as diseased hearts and is not an uncommon manifestation of digitalis intoxication. I have seen many patients with coupled rhythm that remained uncorrected undergo major surgery of all types. Even in those who also had heart disease, the risk was evaluated on the basis of the type of heart lesion and the status of the myocardium. The presence of coupled rhythm was not considered a deterrent if myocardial reserve was judged adequate for surgery. In other words, the presence of extrasystoles is far less significant than the functional efficiency of the myocardium, regardless of the presence of extrasystoles.

Premature auricular extrasystoles, when very frequent, may simulate a slow auricular fibrillation, and often this distinction can be made only by electrocardiography.

SPLIT MITRAL SYSTOLE

A split mitral systole is usually of benign origin but is commonly mistaken for a murmur becauses "it doesn't sound normal." The split sound is due to slight asynchronism between the contractures of the right and left ventricles, which results in separate sounds as they close the mitral and tricuspid valves. A small percentage of cases of split mitral systole are due to bundle branch block. However, in these patients, myocardial tonus is impaired, and there are other evidences of heart disease.

MURMURS

The elicitation of murmurs is a fascinating art. Many times the final diagnosis and even curative therapy of heart disease depends upon this. Aortic insufficiency is positively diagnosed when a soft diastolic aortic murmur is heard. It is usually best elicited by having the patient lean forward and, at the end of

expiration, hold the expired breath. Sometimes it is best heard over the anatomic area of the aortic valve, that is, the third left costal cartilage. Other findings such as cor bovinum, peripheral vascular phenomena, and high pulse pressure are confirmatory, but location of the diastolic murmur is necessary for diagnosis.

The typical machinery murmur of patent ductus arteriosus is another murmur pathognomonic of the lesion. So indicative is this finding of patent ductus arteriosus that surgical correction can usually be recommended on the basis of this finding alone without angiocardiography or cardiac catheterization studies.

The varied murmurs of mitral stenosis present more of a problem in evaluating the degree of stenosis and insufficiency in the average case. For best results, the cardiac surgeon would like always to find a tight mitral stenosis when he opens the heart. If he finds dominant mitral regurgitation, surgery is much less helpful. A fair number of cases diagnosed as predominant mitral stenosis have proved to be dominant mitral insufficiency at surgery. These cases have resulted in some rewriting of the literature on heart sounds in mitral stenosis. This revision is currently being debated by internists and surgeons.

The mitral systolic murmur is probably the most commonly recognized. Many of these are functional and have no significance in heart disease. The functional murmur may usually be distinguished by the following points: (1) It is always systolic, (2) usually grade I, (3) poorly transmitted, and there is (4) no cardiac enlargement.

On the other hand, organic murmurs are harsh—usually from Grade II to Grade VI, are well transmitted to the left axilla, and are accompanied by some degree of cardiac enlargement, especially left ventricular. The harsh systolic murmur of aortic stenosis should always be recognizable by the markedly diminished or absent second aortic sound.

During auscultation, all valvular areas should be examined. When murmurs or adventitious sounds are heard, the physician must not take "big jumps" with the stethoscope but rather move gradually from one valvular area to another.

Evaluation of the second pulmonic heart sound is very important in the diagnosis of heart failure and as a confirmatory finding in mitral stenosis. Until the age of 21 or 22, the second pulmonic sound is naturally louder than the second aortic sound. After this, the pulmonic second sound should recede and the second aortic sound should be louder. In patients above this age bracket, then, an accentuated pulmonic second sound probably has pathologic significance. In hypertensives, as left ventricular failure occurs, the pulmonic second sound exceeds the aortic second sound in intensity. This is an extremely helpful finding in suspected heart failure.

AUSCULTATION OF THE LUNGS

In the detection of left ventricular failure, dependence is placed on eventually finding crepitant rales at the lung bases. These are an indication of low-grade pulmonary edema analogous to the pitting edema of the lower extremities in right heart failure. Basal rales in heart failure always appear first at the right base. This is due to the position of the right hemiazygos vein which positionally does not drain as perfectly when circulation is impaired because of the higher angulation of the right bronchus.

SUMMARY

The importance of physical methods in the diagnosis of cardiac disease, and particularly in certain forms of it, has been studied.

The fact that in cardiac diagnosis the history is often of more value than the physical examination has been pointed out.

Signs and findings during inspection and palpation are described.

Blood pressure, pulse pressure, and heart rate and rhythm have been discussed in relation to diagnosis and prognosis of heart disease.

Auricular fibrillation, extrasystolic arrhythmia, split mitral systole, and murmurs have been discussed.

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ILLINOIS SUPREME COURT INVALIDATES DISCRIMINATORY FINDINGS OF MEDICAL EXAMINING COMMITTEE OF ILLINOIS

In a decision dated April 19, 1955, the Illinois Supreme Court rendered an opinion affirming a decision of the Illinois Appellate Court rendered June 22, 1954, which had set aside an order of the Medical Examining Committee of the Department of Registration and Education of the State of Illinois denying to the Chicago College of Osteopathy approval so that its graduates would be eligible to take the examination for a license to practice medicine in all of its branches in Illinois. An applicant for a license to practice medicine in all of its branches in Illinois under the Medical Practice Act must be a graduate of an approved college in addition to certain other statutory requirements including completion of an approved internship of at least 12 months' duration. The decision of the Illinois Supreme Court set out in full below pertains only to the approval of the Chicago College of Osteopathy as an institution approved for the training of graduates for the full examination given by the Medical Examining Committee. For many years, the school has been approved by the Medical Examining Committee, but only to the extent that its graduates might take a partial examination, not including the subjects of materia medica, therapeutics, surgery, and theory and practice; such graduates have received in the past a license to treat human ailments without the use of drugs or medicines and without operative surgery. The decision of the Illinois Supreme Court holds invalid six findings made by the Medical Examining Committee because such findings were either contrary to the manifest weight of the evidence, violated no rule of the department, or were discriminatory in violation of Section 20 of the Medical Practice Act. This decision is final, and time for the filing of a petition for rehearing before the

Illinois Supreme Court by the Medical Examining Committee has now expired.

ILLINOIS SUPREME COURT

Decided April 19, 1955

Docket No. 33387—Agenda 25—January, 1955.

CHICAGO COLLEGE OF OSTEOPATHY, APPELLEE, V. NOBLE J. PUFFER, DIRECTOR OF THE DEPARTMENT OF REGISTRATION AND EDUCATION, APPELLANT.

5 ILL. 2d—, 126 NE 2d 26

MR. JUSTICE HERSHEY delivered the opinion of the court:

This is an appeal from a decision of the Appellate Court, First District, reversing the superior court of Cook County, which in a proceeding under the Administrative Review Act had affirmed an administrative decision of Noble J. Puffer, Director of the Department of Registration and Education.

On May 31, 1949, the Chicago College of Osteopathy (herein called plaintiff) filed an application with the Department pursuant to the Medical Practice Act (Ill. Rev. Stat. 1949, chap. 91, pars. 1 et seq.) for inspection and approval as a college "reputable and in good standing" for the teaching of students in the treatment of human ailments, so that graduates thereof would be permitted to take the examination for a license to practice medicine in all of its branches in Illinois.

An inspection of the plaintiff college was made by the Medical Examining Committee. After a hearing, the committee filed a report with the Department in which it recommended that the application be denied for failure to meet the statutory requirements and the rules and regulations adopted by the Department for approved medical colleges. The specific reasons assigned for refusing recognition were as follows:

"(1) Rule 5 on page 3, entitled 'Premedical Education, College Preparatory', among other things, provides for a minimum of 16 hours of Chemistry and 8 hours of English, while a review of the catalogue of the Chicago College of Osteopathy shows failure to meet this requirement, in that their minimum requirement is 12 hours of Chemistry and 6 hours of English.

"(2) In Rule 7 on page 4, the Department rules require a minimum of 5119 didactic and laboratory hours for graduation. The said Chicago College of Osteopathy fails to meet that requirement in that the minimum number of hours they require in didactic and laboratory instruction is 4872 hours.

"(3) Under Section 5(a), 'Faculty Requirements', the Department rules require a school to provide at least 6 expert, thoroughly trained professors in the laboratory branches who shall devote their entire time to the college in instruction and research. The catalogue of the Chicago College of Osteopathy shows that they have only 3 men of professorial rank as full-time instructors in basic sciences.

"(4) The Chicago College of Osteopathy also fails to comply with Section 5(b) of the Department rules which is quoted below, since its catalogue shows the names of only 2 licensed physicians and surgeons as members of the faculty: 'The faculty should be composed of graduates of institutions recognized as reputable medical colleges, who have had training in all departments of medicine. Non-medical men should be selected as teachers in medical colleges only under exceptional circumstances, and only when medical men of equal special capacity are not available. The faculty should be organized, each department having as its head professor a physician and surgeon who shall be licensed to practice medicine in all of its branches in the state in which the medical college is located, and who shall have practiced his profession at least ten years, and has devoted the five years next preceding such engagement to the exclusive practice of the specialty in which he is proficient, its associate professor, assistant professor, instructor, etc., each having his particular subjects for the teaching of which he is responsible to the head of the department.'

"(5) The number of anatomy tables provided by the Chicago College of Osteopathy is inadequate for the number of students in the freshmen class, since they have only 12 tables for 68 students. The usual practice followed by approved medical colleges is 4 students to one table.

"(6) Under Section 7, 'Clinical Resources', the Department rules provide that the work of the student at all times shall be under the guidance of a competent instructor who shall hold a license to practice medicine and surgery in all its branches, and since there are only two physicians and surgeons on the faculty of the Chicago College of Osteopathy who are licensed in Illinois, it is impossible for them to supervise all students in the college."

The Department, in denying the application, approved and adopted the report and recommendation of the committee.

The plaintiff then petitioned for a rehearing, and upon rehearing held August 10, 1950, offered certain evidence. The committee again recommended the denial of the application, and the Department adopted that recommendation. This final administrative determination of the Department was based upon both findings of the committee, quoted above, and the evidence adduced upon rehearing in opposition to the findings.

The superior court of Cook County affirmed the decision of the Department, and the Appellate Court reversed and remanded the cause with directions to refer the matter to the Department for the entry of an order approving plaintiff's application. *(Chicago College of Osteopathy v. Puffer,* 3 Ill. App. 2d 69.) We allowed the Department's petition for leave to appeal.

The Appellate Court, as a basis for remanding the case to the Department for the entry of an order approving the plaintiff's application, was of the opinion that the plaintiff had substantially complied with the requirements in accordance with the standards which the Department has in practice applied. The Department contends that the Appellate Court wrongfully substituted its own judgment for that of the Department, thereby usurping the function of said administrative agency.

The position of the Department may be summarized as follows: The legislature has delegated to the Department, acting upon a report of the examining committee, the function of approving and accrediting colleges whose graduates may be allowed to take an examination for a license to practice medicine in all its branches in Illinois. The Department properly exercised said statutory power in this case and its action in denying the application of the plaintiff college was in accord with the law and the evidence. Since the plaintiff admittedly failed to satisfy the rules of the Department in the particulars stated, and since it is not argued that said rules establish unconstitutional requirements, the judgment of the superior court of Cook County should be affirmed.

The plaintiff, in urging us to affirm the Appellate Court, contends the record in this case fully supports the conclusion reached by that court, since the action of the Department in denying plaintiff's application constituted a violation of sections 19 and 20 of the Medical Practice Act. The Department, it is argued, violated section 20 by requiring the plaintiff to meet higher standards for approval than the standards generally required of colleges which grant the degree of Doctor of Medicine.

The present Medical Practice Act was passed in 1923, and its constitutionality was upheld in *People v. Witte,* 315 Ill. 282. The legislative plan approved in that case divides licenses, so far as physicians are concerned, into two classes—one which confers the right to practice medicine in all of its branches, and the other to treat human ailments without the use of drugs or medicines and without operative surgery. (Ill. Rev. Stat. 1949, chap. 91, par. II.) A similar classification is followed regarding minimum standards of professional education (Ill. Rev. Stat. 1949, chap. 91, par. 5) and the scope of examinations. Ill. Rev. Stat. 1949, chap. 91, pars. 8, 9.

Section 19 of the act vests with the Department the power and duty to accredit professional colleges whose graduates may be allowed to take an examination for one or the other type of license. This section empowers the Department, among other things, "To make rules for establishing reasonable minimum standards of educational requirements to be observed by medical colleges, or by any professional school, college, or institution teaching any system or method of treating human ailments, or by colleges of midwifery, and to determine the reputability and good standing of all schools, colleges, and institutions now, heretofore, or hereafter existing." (See also Ill. Rev. Stat. 1949, chap. 127, par. 60.) The Medical Practice Act further provides that none of the functions, powers and duties enumerated therein shall be exercised by the Department except upon the action and report in writing of the Medical Examining Committee. Ill. Rev. Stat. 1949, chap. 91, par. 16b-I; see also chap. 127, par 60a.

It will be noted that section 19 makes it the duty of the Department to adopt reasonable standards of educational requirements to be observed by professional schools. Despite this clear legislative direction, the Department for a period of twenty years failed to adopt any such rules, and during that period no student who did not hold the degree of Doctor of Medicine was permitted to take the examination for an unlimited license. However, rules were adopted in 1943, but only after an original mandamus suit was begun by the plaintiff college asking this court to order the Department to take such action. The plaintiff thereafter made an effort to comply with the established rules, expending some $500,000 to this end, and in May, 1949, submitted its application for inspection and approval.

We turn next to section 20 of the act, which in effect prohibits discrimination against osteopaths and others similarly situated. This section, also contained in the Medical Practice Act of 1923, reads as follows: "The provisions of this Act shall not be so construed as to discriminate against any system or method of treating human ailments, or against any medical college, or any professional school, college or institution teaching any system or method of treating human ailments, on account of any such system or method which may be taught or emphasized in such medical college, or in such professional school, college or institution."

Section 20 was an affirmative declaration by the legislature of what this court said was required by the constitution in the administration of a medical practice act. Provisions of prior medical practice acts which purported to authorize the very discrimination which the present act prohibited had been invalidated. In *People v. Schaeffer,* 310 Ill. 574, we declared the Medical Practice Act of 1899 unconstitutional in its application to osteopaths, for the reason that it provided that no applicant could take an examination for a license to practice medicine and surgery in all branches unless he was a graduate of a medical college, as distinguished from an osteopathic college. This court said at page 583: "The very great prejudice existing among many physicians of the medical schools against the osteopaths, and of the osteopaths against those of the medical schools, is well known. This statute recognizes both systems as meritorious because it allows both to treat human ailments according to their system, and it discriminates against the osteopath and seems to place the examinations of osteopaths to practice osteopathy entirely at the will and discretion of a medical board, as no one other than those educated in the medical system are qualified, under the act, to conduct the examinations provided for by it. This statute therefore tends to deprive the osteopaths of their constitutional right to practice surgery, who are, so far as this record shows, just as efficient and as well prepared by college and hospital training to practice surgery as are the physicians of the medical schools. The act is therefore void as to such physicians so deprived." See also *People v. Graham,* 311 Ill. 92.

The plaintiff college, which is located in Chicago, has been approved by the Department so that its graduates may take the examination for a limited license (i.e., a license to treat human ailments without the use of drugs or medicines and without operative surgery), but in the instant application it seeks approval so that its graduates may take the examination for a license to practice medicine in all its branches. The examina-

tion for the limited license is the same as that given to applicants for a full license except the latter are also examined on materia medica, therapeutics, surgery, obstetrics, and theory and practice. Ill. Rev. Stat. 1949, chap. 91, par. 9.

The evidence presented to the Department shows the following: The plaintiff was incorporated as a not-for-profit educational institution in Illinois in 1913, and since 1918 has had one block of land between Fifty-second and Fifty-third streets in Chicago on which are located buildings containing class rooms, laboratories, a library, an out-patient department and a one-hundred bed hospital. It has affiliated with it for teaching purposes the Detroit Osteopathic Hospital in Detroit, Michigan. Students in their senior year spend three months at this hospital where they receive additional instruction in surgery.

It is further shown that the plaintiff employs a faculty of sixty-four people, who are trained in all branches of medicine and surgery. It operates on an annual budget of $700,000, and at the time of the hearing its student enrollment was 214. Its educational program has been accepted by every agency, both Federal and State, to which it has applied, with the exception of the Department in Illinois, and in Illinois its graduates are permitted to take the examinations for a license to treat human ailments without the use of drugs and without operative surgery and for a license to practice obstetrics. It teaches materia medica, therapeutics, surgery, obstetrics, and theory and practice, and its instruction in these subjects parallels that given in approved medical institutions. Graduates of the plaintiff college are permitted by the following States and other jurisdictions to take examinations for an unlimited license to practice medicine: Indiana, New Jersey, New York, Ohio, Wisconsin, Colorado, Connecticut, Delaware, District of Columbia, Kentucky, New Hampshire, Oregon, Rhode Island, South Dakota, Texas, Virginia, Wyoming, Arizona, Florida, Hawaii, Iowa, Maine, Michigan, Missouri, Nevada, New Mexico, Oklahoma, Pennsylvania, Tennessee, Utah, Vermont, Washington, and West Virginia. Of the 242 students graduated by plaintiff in the last ten years, 196 went to States where they were permitted to take examinations for an unlimited license and all of them passed, eighty per cent passing on the first examination.

We now come to the issue in the case, namely, whether the Department's denial of plaintiff's application was a proper exercise of its administrative function. Of all the complex and stringent requirements and standards established by departmental rules and regulations, it was found that the plaintiff failed to comply in six particulars. These findings of the Department, set out in full above, must be considered in detail with a view to determining: (1) Is the rule reasonable? (2) Is the finding supported by the evidence? and (3) Does the rule as applied amount to a violation of section 20 of the Medical Practice Act?

Finding No. 1 recited a violation of a rule providing for a minimum of 16 hours of Chemistry and 8 hours of English for admission to the school. It is conceded that at the time of the inspection the plaintiff required 12 hours of Chemistry and 6 hours of English.

Plaintiff insists, however, that this rule is not enforced by the Department against medical colleges which grant the degree of Doctor of Medicine and which have received the Department's approval. The results of a study made of catalogues of 45 of the 80 colleges approved by the Department disclosed that 27 of them required only 6 hours of English for admission, including Northwestern University (1948-9), University of Illinois (1948-9), and St. Louis University (1948-9). Moreover, it was established that 17 of these approved colleges required less than 16 hours of Chemistry, including New York University (1947-8), University of Nebraska (1947-8), and Western Reserve University (1948-9), each of which listed an admission requirement of 12 hours. Also introduced in evidence was a booklet published in 1950 by the Association of American Medical Colleges which showed that 49 of the approved schools then had admission standards violative of this rule.

Finding No. 2 recited a violation of a rule requiring a minimum of 5119 didactic and laboratory hours for graduation. The plaintiff's requirement in this regard was 4872 didactic and laboratory hours.

However, of the same 45 colleges referred to above, it was shown that 21 listed their hours in their catalogues as less than the number specified in this rule. For example, Harvard Medical School (1946-8) listed 4208, Indiana University (1948-9) listed 4716, Northwestern University (1948-9) listed 4707, Ohio State University (1948-9) listed 4836, and the University of Illinois (1949-50) listed 4629.

Finding No. 3 recited a violation of a rule requiring a school to provide at least 6 expert, thoroughly trained professors in the laboratory branches who shall devote their entire time to the college in instruction and research. The Department based this violation on a finding that the catalogue of the plaintiff college showed that it had only 3 men of professorial rank as full time instructors in basic sciences.

We believe this finding is against the manifest weight of the evidence, for apart from the listing in the catalogue, the undisputed evidence establishes that at the time of the inspection there were 6 staff members who qualified under this rule. It is true that of the six, two were designated by the college as assistant professors, but we see no justification for reading the word "professors" as indicating the academic rank of full professor. Academic rank is a matter solely between the school and the teacher, and there is no necessary correlation between academic rank and teaching ability. In any event, the record shows without contradiction that on the staff of the plaintiff college were 6 expert, thoroughly trained teachers in the laboratory branches who devoted their entire time to the college of instruction and research.

Finding No. 4 recited a failure to comply with a rule of the Department pertaining to faculty requirements. This rule, quoted in full above, contains four requirements: (1) The faculty should be composed of graduates of institutions recognized as reputable medical colleges who have had training in all departments of medicine, with a provision that nonmedical men should be selected as teachers in medical colleges only under exceptional circumstances and only when medical men of equal special capacity are not available. (2) Heads of departments must be licensed to practice medicine in all its branches in the State in which the medical college is located. (3) Heads of departments must have practiced their profession for ten years, five of which must be devoted exclusively to their particular specialty. (4) Departments must be manned by proper associates and assistants.

It is with reference to this finding that the most crucial question in the case is raised and about which extended comment must be made.

In the finding, the rule is said to be violated because the catalogue of the plaintiff college lists the names of only two licensed physicians and surgeons as members of the faculty. The Department must be referring to the second of the enumerated requirements, which states that each department head shall be licensed to practice medicine in all of its branches in the State where the medical college is located. That such is the proper reference is borne out by argument of counsel for the Department who say the plaintiff violated this rule by reason of the fact only two staff members hold an unlimited license in Illinois.

The three major departments in the plaintiff college are the Department of Surgery, the Department of the Practice of Osteopathy (which corresponds to the Department of Medicine in schools which grant the degree Doctor of Medicine) and the Department of Obstetrics and Gynecology. The head of the Department of Surgery does have an unlimited Illinois license, but the heads of the other departments do not. However, the head of the Department of the Practice of Osteopathy holds licenses to practice medicine in all of its branches in California, Michigan, and Missouri. The head of the Department of Obstetrics and Gynecology, while he does not have an unlimited Illinois license, is licensed to practice obstetrics in Illinois. The other departments of the college are the Department of Anatomy, the Department of Bacteriology and Public Health, the Department of Pathology and the Department of Physiology and Pharmacology.

This requirement, on its face, does not appear to be arbitrary or unreasonable. To require the head of a department to hold a license in the State where the college is located may seem to be a standard reasonably calculated to help insure the quality of the faculty. For it could be argued that the Department then has the satisfaction of knowing that said department heads are qualified physicians in good standing in the State where they teach and are subject, the same as other physicians practicing within the States, to supervision and control by a board or department charged with responsibility of enforcing the State's policy relative to the practice of medicine in all its branches.

But this requirement was established by the Department despite an explicit statement by the legislature that there shall be no discrimination against colleges by reason of any system or method of treating human ailments which may be taught or emphasized. And when the facts of this record are considered with respect to this statutory provision, especially in the light of the legislative history discussed above, we are convinced that the requirement is unreasonable and discriminatory as applied to the plaintiff college.

First, the plaintiff college has been approved by the Department so that its graduates may take an examination for a limited license. The examination for a limited license and for an unlimited license is identical with the exception that applicants for an unlimited license are also questioned on materia medica, therapeutics, surgery, obstetrics, and theory and practice. The undisputed evidence in this record is that the instruction given at the plaintiff college on these subjects is equal to that given in colleges approved by the Department, considered both quantitatively (hours devoted to subject, textbooks used, etc.) and qualitatively (teaching ability of the faculty, etc.). Indeed, apart from the failure to hold unlimited licenses in Illinois, the background, education and ability of the heads of departments, as well as of the remainder of the faculty, are not said to be or demonstrated to be in any way inferior to those of men who staff colleges which have received departmental approval.

Second, the plaintiff's educational program has been fully approved by every agency, both Federal and State, to which it has applied, with the exception of the Department in Illinois. Its graduates are permitted to take examinations for an unlimited license to practice medicine in all branches in 31 States plus the District of Columbia and Hawaii.

Third, as already related, the legislative history in Illinois regarding the osteopathic profession is replete with examples of discrimination. To its credit, however, the legislature enacted section 20 of the act in an effort to insure just treatment for osteopaths and others similarly situated, but this record shows that the Department has still failed to desist from that type of practice which was condemned by this court and the legislature as long ago as 1923. One instance from the evidence will illustrate. One of the plaintiff's staff, who holds an unlimited license to practice medicine in Colorado, made an application for a full license in Illinois under the reciprocity provisions of the Illinois act. The Department, however, refused his application, and in a letter to him dated July 21, 1950, said "the Illinois Medical Practice Act does not provide for the acceptance of a diploma of graduation from an osteopathic college as a basis for issuance of a medical license either by reciprocity or examination." This shows the difficulty encountered by the plaintiff college in attempting to meet the requirement under consideration, and the Department's attitude further indicates that the purpose of the rule is to prevent the plaintiff college (or any osteopathic college for that matter) from qualifying rather than to safeguard the public from incompetent members of the healing arts profession.

Significantly, the list of the 80 colleges approved by the Department is identical with those approved by the American Medical Association, which regards the practice of osteopathy as an unscientific, cultist practice. The entire membership of the Medical Examining Committee is composed of men who hold the degree of Doctor of Medicine and are members of the American Medical Association.

Finally, here again the Department has made apparent exceptions in the enforcement of the rule, for the evidence shows that there are several of the 80 approved colleges which have some heads of departments who are not licensed to practice medicine in all branches in the State where the college is located or even have any type of healing art degree, be it a Doctor of Medicine (M.D.) or a Doctor of Osteopathy (D.O.).

Finding No. 5 recited that the number of anatomy tables provided by the plaintiff college is inadequate for the number of students in the freshman class. It is said the plaintiff has only 12 tables for 68 students, and the usual practice followed by approved medical colleges is four students to one table. This finding does not state the violation of a rule or regulation of the Department; therefore, disapproval of the college could not be based, even in part, upon the ground stated. The evidence shows that there is nothing in the rules specifying the number of tables for dissection, but there is a requirement that each student must dissect the lateral half of a cadaver, which means two students to a cadaver. However, because of the shortage of cadavers none of the medical schools in Chicago can comply with this rule, and the plaintiff receives cadavers by allocation from an association of which a representative of the plaintiff college is a member, in the same ratio to the number of students as the other approved medical colleges in Chicago.

Finding No. 6 recited a failure to comply with a rule of the Department providing that the work of a student at all times shall be under the guidance of a competent instructor who shall hold a license to practice medicine and surgery in all its branches. The Department states that it is impossible for the plaintiff college to meet this requirement since there are only two physicians and surgeons on the faculty who are licensed in Illinois. But the rule does not say the supervision must be under one who is licensed in Illinois in all branches. Were it to so provide, it would be subject to the same objections pointed out with regard to finding No. 4. But since the rule does not require the supervising instructor to be licensed in Illinois, it is sufficient to point out that plaintiff college literally complies with this rule. Many of the staff are licensed in other jurisdictions to practice medicine in all branches, and so far as the record is concerned, it is undisputed that the supervision of students is adequate and that each is, in fact, under the guidance of a competent instructor who is trained in all branches of medicine and who holds a license to practice his art in all its branches.

Summarizing, it is the opinion of this court that findings No. 3 and No. 6 are contrary to the manifest weight of the evidence and must be set aside. Finding No. 5 does not state the violation of a rule of the Department and is, therefore, set aside. Findings No. 1, No. 2, and No. 4 are nullified because the uncontradicted evidence in the record demonstrates that the requirements stated as applied to this plaintiff are violative of section 20 of the Medical Practice Act.

The grounds for denying recognition to the plaintiff college are all invalid; therefore, at the time of said order by the Department the plaintiff was qualified as a college reputable and in good standing whose graduates are eligible to take the examination for a license to practice medicine in all its branches in Illinois. The judgment of the Appellate Court is affirmed and the cause is remanded to the Department with directions to expeditiously determine whether any changes have occurred subsequent to that time which justify withholding approval. Said determination is ordered to be made with the object of effectuating the manifest intention of the legislature as elaborated in this opinion.

Judgment affirmed, and cause remanded, with directions.

Department of Public Relations

CHESTER D. SWOPE, D.O.
Chairman
Washington

FEDERAL AID TO EDUCATION

STATEMENT
OF
AMERICAN OSTEOPATHIC ASSOCIATION
BY
J. S. DENSLOW, D.O.
SUBCOMMITTEE ON HEALTH
SENATE COMMITTEE ON LABOR AND PUBLIC WELFARE
RE: S. 1323
FEDERAL AID TO MEDICAL EDUCATION
May 6, 1955

Mr. Chairman and Members of the Subcommittee, I am J. S. Denslow, D.O., Director of Research Affairs, Kirksville College of Osteopathy and Surgery, and Secretary-Treasurer of the American Association of Osteopathic Colleges, and appear here representing the American Osteopathic Association.

My background in research may be objectively illustrated in a listing of contributions to nonosteopathic scientific literature, which I ask may be inserted at the end of my remarks. The American Association of Osteopathic Colleges comprises all existing colleges of osteopathy and surgery, each of which is approved by the American Osteopathic Association. The American Osteopathic Association has a membership of approximately 9,000 osteopathic physicians or surgeons out of a total of some 12,000 who are licensed and practicing in all the States of the United States.

We very much appreciate the opportunity of expressing our views on S. 1323, cited as the "Medical Educational Facilities Construction Act of 1955."

S. 1323 recognizes the need for Federal assistance to medical schools for construction of facilities urgently needed to enable the schools to carry out their responsibility for the quality and number of physicians being trained, for the education of teachers and research workers, and for the conduct of needed research.

Osteopathic physicians are licensed and practicing in all the States. Approximately 90 per cent of the profession is located in States (three-fourths of the States) granting licenses to engage in the general practice of the healing art, including major surgery, drug therapy, and obstetrics. Some 12,000 osteopathic physicians or surgeons are engaged in general or specialty practice. As stated in the Guidance Leaflet on Osteopathy, prepared by the U. S. Office of Education, such specialties include: anesthesiology, diagnostic roentgenology, internal medicine, neurology and neurosurgery, obstetrics, obstetrics and gynecology, obstetrical and gynecological surgery, ophthalmology and otorhinolaryngology, orthopedic surgery, pathology, pediatrics, psychiatry, radiology, roentgenology, surgery and urological surgery.

In excess of three hundred hospitals are staffed by osteopathic physicians or surgeons. Eighty-five hospitals are approved for intern or residency training.

There are six colleges of osteopathy and surgery, all nonprofit, tax-exempt, approved by the American Osteopathic Association, the Veterans Administration, and the Public Health Service.

Chicago College of Osteopathy, Chicago, Illinois, Established 1902.

College of Osteopathic Physicians and Surgeons, Los Angeles, California, Established July 14, 1896.

Des Moines Still College of Osteopathy and Surgery, Des Moines, Iowa, Established June 8, 1898.

Kansas City College of Osteopathy and Surgery, Kansas City, Missouri, Established 1916.

Kirksville College of Osteopathy and Surgery, Kirksville, Missouri, Established 1892.

Philadelphia College of Osteopathy, Philadelphia, Pennsylvania, Established January 24, 1899.

Senator BENDER, of Ohio. Do any of them now, or have any of them in the past, received any Federal or State aid?

Dr. DENSLOW. Yes, I will get to that, Senator Bender, shortly.

In the Fall of 1954, 1,867 students were enrolled in the osteopathic colleges. The freshman class (487) received preprofessional training in colleges and universities in 38 States and the District of Columbia. These matriculants had preprofessional college training as follows: 72 per cent had baccalaureate or advanced degrees, 98 per cent had 3 or more years, and the remainder had 2 or 2-plus years of preprofessional college training. A minimum of 3 years of preprofessional training is required for the class entering in 1955. The professional course is 4 years. There were 449 graduates in 1954. During the past 5 years an average of 442 have graduated. Most graduates take 1 or more years of intern-training.

The average tuition is $700 a year. The student in paying this yearly tuition of $700 is paying less than one-third of the actual cost of his education. The average osteopathic college spends approximately $2,400 per year per student in conducting the type of educational program which the profession has insisted upon.

The difference between undergraduate tuition and fees and the school budgets is made up as nearly as possible by indebtedness, hospital, clinic and graduate course fees, grants, such as cancer and heart teaching grants from the National Institutes of Health, and gifts.

This is the comment I was going to make. These grants are both for teaching in the field of heart disease and cancer.

Senator BENDER. These are grants from public . . .

Dr. DENSLOW. From the Public Health Service, the National Institutes of Health. In addition to these teaching

grants we have in one institution been operating under research grants from the Public Health Service since 1947.

Senator BENDER. These bills cover a different field entirely. These are bills providing for construction.

Dr. DENSLOW. That is right, sir.

What I have been trying to point out is that—or will try to point out—that if funds for construction are made available, the money that is freed in the college budget can be used to improve and extend the operational procedure.

In 1943, a continuing Osteopathic Progress Fund (nonprofit tax-exempt) was established, through which the individual members of the profession and others make annual contributions to the colleges. These contributions total in excess of $5 million to date.

Senator BENDER. Just a brief interruption. You state that your college is in Kirksville, Missouri. Is that a State school?

Dr. DENSLOW. No, sir. All the osteopathic colleges are private, nonprofit institutions.

A survey of the demonstrable needs of the colleges for additional teaching space, which was conducted last November, showed needs in excess of 182,500 square feet at an estimated cost of $3,535,000, and none of the colleges has funds to proceed with the construction.

These figures do not include necessary additional space for expanding research programs. Although the Bureau of Research of the American Osteopathic Association had meritorious applications for research projects totalling nearly $100,000 for 1953-1954, and $100,000 for 1954-1955, grants were able to be made in the amounts of $52,750 and $66,100 for the 2 years, respectively. At my own college in Kirksville, Missouri, we are optimistically proceeding with preliminary plans for the construction of urgently needed additional facilities to house our research program, but actual construction of the facilities will be impossible in the current financial situation of the college without major Government assistance. The research carried on at the college has been made possible by grants from the American Osteopathic Association, the U. S. Public Health Service, the Bureau of Naval Research, and various trust funds, and individual contributions.

The problem of space for research programs was succinctly stated in the July, 1954, annual report of the Bureau of Research of the Association as follows:

> One of the major problems in establishing research programs in osteopathic institutions is lack of space that can be assigned for this purpose. There is also the problem of accumulation of the basic equipment to convert such space, when found, into a research laboratory. It is permissible to expend part of grant monies for major scientific equipment, but it is not permissible to make plant alterations or buy and install basic laboratory furniture and utilities. If the A.O.A. did not have to appropriate large amounts for the major support of the research programs, it could be in a position to assist the colleges in setting up essential laboratory facilities that would help attract assistance from outside granting agencies.

The cancer and heart teaching grants provided to the osteopathic colleges by the National Cancer Institute and the National Heart Institute not only help tremendously to emphasize the impact of cancer and heart disease and the methods of detecting and dealing with them, but help also to kindle and channel in their direction the investigative interest of students who have aptitude for scientific research. Stimulation of interest and discovery of aptitude for research must be followed by training in scientific methods. Such training requires not only qualified supervision, but adequate laboratory space and equipment.

Mr. Chairman, participation in the program under this bill would enable the osteopathic colleges to improve and expand their teaching and research facilities and increase to some extent their output of physicians. However, *the bill as written makes no provision for schools training physicians who are doctors of osteopathy.* Therefore, the following amendments which are designed to include osteopathic schools are respectfully submitted:

1. Page 4, lines 5 and 6, strike "the degree of doctor of medicine" and substitute "a degree of doctor of medicine or osteopathy."

2. Page 4, lines 7, 8, and 9, strike the words "after he has obtained the advice and recommendation of the Federal Council on Medical Education Facilities created under section 703."

3. Page 4, line 21, strike the period after the word "health" and insert a comma and the words "including persons active in each of the fields of professional education concerned."

The Federal Council on Medical Education Facilities, which is provided for in the bill, and which should have representation as proposed in our amendment, should serve as adviser to the Surgeon General in connection with grants and with respect to regulations relating thereto.

Our proposed amendments follow the form adopted by the Senate Committee on Labor and Public Welfare in previous projected legislation. I refer to S. 1453 of the 81st Congress, and S. 337 of the 82nd Congress, both of which provided in part for construction grants for schools of osteopathy.

The War Manpower Commission in 1942 certified that the practice of osteopathy was vital to war production and the support of the war effort, and an activity the maintenance of which was necessary to the health, safety and welfare of the Nation, and that there were "serious shortages" of persons trained, qualified, or skilled to engage in the critical occupation of osteopathy (Selective Service Occupational Bulletin No. 41, December 14, 1942). In 1950, the U. S. Department of Labor included osteopathy in its list of critical occupations. It has continued so listed, including the revised current list of critical occupations issued March 2, 1955. The list is compiled on the recommendations of the Interagency Advisory Committee on Essential Activities and Critical Occupations. The Committee consists of representatives of the Departments of Defense, the Interior, Agriculture, as well as Commerce and Labor, and the Selective Service System. The criteria applied require that occupations listed are those in which there is an "over-all shortage," and in which the "occupation is indispensable" to the functioning of the activity in which it occurs.

We extend our deep appreciation to the Chairman and the Committee for their leadership in this extremely important matter.

We feel the basic approach of providing for the constructing of teaching and research facilities on a matching basis is a practical and very worth-while method.

CHAIRMAN. Doctor, thank you very much for coming.

CONTRIBUTIONS TO NONOSTEOPATHIC LITERATURE BY J. S. DENSLOW, D.O.

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2. Denslow, J. S., and C. C. Hassett. The central excitatory state associated with postural abnormalities. Journal of Neurophysiology, 5:393-402, 1942.
3. Denslow, J. S., and C. C. Hassett. The polyphasic action currents of the motor unit complex. American Journal of Physiology, 139:4, 1943.
4. Denslow, J. S. An analysis of the variability of spinal reflex thresholds. Journal of Neurophysiology, 7:207-216, 1944.
5. Denslow, J. S., and N. L. Cuthbert. Electrode efficiency and subject positioning in electromyography. Proceedings of the Society of Experimental Biology and Medicine, 58:191-193, 1945.
6. Denslow, J. S., I. M. Korr, and A. D. Krems. Quantitative studies of chronic facilitation in human motoneuron pools. American Journal of Physiology, 105:2, 1947.
7. Denslow, J. S. Double discharges in human motor units. Federation Proceedings, 6:1, 1947.
8. Denslow, J. S., and David M. Graham-Service. The spread of muscle action potentials from active to inactive area. Federation Proceedings, 7:1, 1948.
9. Denslow, J. S. Double discharges in human motor units. Journal of Neurophysiology, 11:209-216, 1948.
10. Denslow, J. S., and Olwen R. Gutensohn. Distribution of muscle fibers in a single motor unit. Federation Proceedings, 9:1, March 1950.
11. Denslow, J. S., and Olwen R. Gutensohn. Neuromuscular organization of single motor units. Federation Proceedings, 10:1, March 1951.

RENEW FEDERAL NARCOTIC AND MARIHUANA PERMITS

Osteopathic physicians and osteopathic colleges or research laboratories must renew their Federal narcotic permits every year before July 1. Failure to do so entails severe penalties.

On or before July 1 each osteopathic physician who wishes to dispense, prescribe, or administer marihuana in his practice must, under the provisions of the Federal Marihuana Tax Act, pay a $1.00 annual tax and register or reregister with the Director of Internal Revenue for each of the districts in which he practices.

D.O.'S ATTEND NATIONAL POLIO VACCINE MEETING

The American Osteopathic Association was represented by Chester D. Swope, D.O., Chairman of the Department of Public Relations, and R. C. McCaughan, D.O., Executive Secretary of the Association, at the April 22, 1955, Scientific and Technical Conference on the Distribution of the Polio Vaccine, which was called to meet in Washington at the request of the President. The invitation and the press notice of organizations invited are reproduced below.

WESTERN UNION TELEGRAM

1955 APR 19

DR CHESTER D SWOPE, CHAIRMAN DEPT OF PUBLIC RELATIONS
OSTEOPATHIC ASSN FARRAGUT MEDICAL BLDG, WASH DC
IN CONFORMITY WITH THE PRESIDENTS WISHES WOULD APPRECIATE HAVING NOT MORE THAN TWO REPRESENTATIVES OF YOUR ORGANIZATION ATTEND MEETING WITH MEMBERS OF MEDICAL, PUBLIC HEALTH PROFESSIONS AND INDUSTRY TO DISCUSS QUESTION OF EQUITABLE DISTRIBUTION SALK VACCINE, ROOM 5051 HEW BUILDING, WASHINGTON DC NINE-THIRTY AM, APRIL 22, HOPE YOU WILL ATTEND PERSONALLY. WOULD APPRECIATE TELEGRAPHIC REPLY WITH NAMES OF DESIGNATED REPRESENTATIVES.
OVETA CULP HOBBY, SECRETARY DEPT HEALTH EDUCATION & WELFARE

U. S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
OFFICE OF THE SECRETARY
WASHINGTON 25, D.C.

FOR IMMEDIATE RELEASE
Thursday, April 21, 1955 HEW—C8

Representatives from 25 groups and organizations have been invited to attend a technical and scientific conference tomorrow, April 22, on the availability of the Salk polio vaccine, Secretary Oveta Culp Hobby, U. S. Department of Health, Education, and Welfare, said today.

Invitations were also extended to the Chairman and ranking minority member of the Senate Committee on Labor and Public Welfare and the House Committee on Interstate and Foreign Commerce.

The conference was called in accordance with the President's directive to the Secretary to survey and report to him on the best means of assuring an equitable distribution of the vaccine.

Secretary Hobby described the conference as an initial step in gathering information on which to base her report to the President.

The organizations and groups which were invited represent the health and medical professions, and pharmaceutical and drug industries, and other interested professional and State groups. Following is a list of the invited organizations: American Academy of General Practice; American Academy of Pediatrics; American Drug Manufacturers Association; American Hospital Association; American Medical Association; American Osteopathic Association; American Pharmaceutical Association; American Pharmaceutical Manufacturers Association; American Public Health Association; American School Health Association; American Surgical Trade Association; Association of State and Territorial Health Officers; Cutter Laboratories; Eli Lilly & Company; Hospital Industries Association; Manufacturers Surgical Trade Association; National Association of Chain Drug Stores; National Association of Retail Druggists; National Foundation for Infantile Paralysis; National Medical Association; National Wholesale Druggists Association; Parke-Davis & Company; Pittman-Moore Co.; Sharp & Dohme; Wyeth Laboratories, Inc.

Also in attendance will be officials from the Departments of State, Defense, and Commerce, the Office of Defense Mobilization, the Public Health Service, the Social Security Administration, the Children's Bureau, the Food and Drug Administration, the Office of Education, and the Office of the Secretary of the Department of Health, Education, and Welfare. Chairman of the meeting will be Dr. Chester S. Keefer, Special Assistant to the Secretary for Health and Medical Affairs.

Current Literature

VAGINAL HYSTERECTOMY FOR UTERINE PROLAPSE

A modified Spaulding-Richardson operation to correct uterine prolapse avoids many of the complications of classic vaginal hysterectomy, according to William H. Mast, M.D., who reports his use of the technic on thirty-seven patients with incomplete, and fifteen others with complete, prolapse in *Surgery, Gynecology and Obstetrics,* March, 1955.

The Spaulding-Richardson operation entails cervical amputation, removal of the corpus, and retention of the isthmic portion of the uterus for purposes of interposition. The modification consists of removing the corpus *before* the cervix is amputated, to permit continuous cervical traction and ensure better exposure. Dividing the description of the technic into two stages for the sake of clarity, the author provides a step-by-step account of the procedure.

Important considerations in choosing this technic are the patient's age, the likelihood of future pregnancy, and the extent and nature of the prolapse. Radical technics are as a rule avoided in the younger patient. The successful outcome of this operation depends on careful selection of the patients and on meticulous adherence to the technic. Its use is contraindicated when adenocarcinoma is detected in the fundus or body of the uterus, in the event of either cervical carcinoma or cancer in situ, and if large ovarian cysts or solid tumors and intraligamentous cysts exist. Previous pelvic surgery does not always preclude its use, but extreme senile uterine atrophy does. Large uterine leiomyomas, particularly if they are wedged in the pelvis or situated beneath the bladder, would inhibit adequate exposure of the vital structures as well as the proper control of bleeding. Procidentia, especially in the older woman, would call for some form of uterovaginal extirpation. Cardiovascular or renal disease, vaginal ulcers, and diseases and infections of the genitourinary tract are treated preoperatively, and local infection can be reduced postoperatively by intravaginal therapy with antibiotics.

None of the fifty-two patients in this series died as a result of the operation. Of the four who experienced shock, two had cyclopropane hypercapnia, since avoided by proper aeration of the lungs postoperatively. No final solution has been found for preventing urinary complications, and twenty-four patients required catheterization after the Foley catheter was removed. Vaginal bleeding was moderate in twenty-two cases, severe in one. No evidence of internal bleeding, fistulas, or embolic pneumonia were noted. Postoperative menstrual bleeding has been scanty and short-lived except among eight of the younger women who have continued to have regular, though scanty, cycles. In no instance has cystocele, urethrocele, or prolapse of the vault recurred.

THE PRESENT-DAY MANAGEMENT OF ARTHRITIS

The management of arthritis is discussed in the light of its evolution during the past 30 years by Charles Ragan, M.D., in the *Journal of Chronic Diseases,* March, 1955.

An important contribution of this period has been the differentiation of rheumatoid arthritis from degenerative joint disease. Its management depends on the expected life span of the patient, how crippling the disease may become, and the extent to which it will derange his economic and social life. In the early stages, watchful waiting and the use of mild antiphlogistics give the physician time to become acquainted with his patient, to guide him toward a more restful way of life,

and permit spontaneous remission to take place. The advantages of withholding drastic therapy are discussed, and circumstances requiring speedy measures are outlined. Syndromes to be differentiated from rheumatoid arthritis are listed, and the signs and symptoms of such arthritis are outlined. Adequate study of the natural history of the disease and the proper evaluation of therapy require a simple specific diagnostic test, uniform diagnostic criteria, and a method for evaluating its progression.

The cause of rheumatoid arthritis is unknown, and no means exist for terminating the disease. Rest will reduce inflammation, and symptoms can be controlled by empirical measures. Most important to its management are time, patience, and rapport and honesty between the physician and the patient.

Pain influences all of the patient's activities, often even limiting efforts at rehabilitation. An effort must be made to control the pain, but the choice of method or agent will depend on how severe and rapidly progressing the disease is, on the social and economic status of the patient, and on the availability of care. The alternatives to moderately decreased activity all have their hazards and these are discussed. When irreversible deformities exist, the patient must slacken his pace and be made as comfortable as possible, for he can no longer be restored to full function.

CLINICAL ASPECTS OF THE MAJOR PORPHYRINOPATHIES

In a clinic on porphyrinuria and porphyrinorrhea, the clinical manifestations of diseases disturbing porphyrin pigment metabolism are discussed by Robert M. Kark, F.R.C.P., whose report was published in *The Medical Clinics of North America,* January, 1955.

The chemical composition of the porphyrin pigments, which are found in practically every cell in all forms of life, are outlined, and the biosynthesis of porphyrins and heme compounds in man are schematically charted on the basis of information obtained during a decade of investigation. It is known that large amounts of Type III porphyrin are synthesized in the healthy body to provide prosthetic groups for the respiratory enzymes, and that the basic action of the heme compounds is to supply the solar energy stored in food for the metabolic needs of the cell. The major porphyrinopathies, constituting a small group of disorders with striking signs and symptoms related directly to defects in the metabolism of pigments, are clinically classified. The minor porphyrinopathies, constituting a much larger group of diseases but producing no clinically important manifestations, are also reviewed.

The genetically determined aberration of pigment metabolism known as congenital porphyria is rare, but its pathogenesis, its clinical manifestations, and its treatment are discussed, and an illustrative case is presented. Intermittent porphyria, problems involved in its diagnosis, its clinical features, the pattern of unpredictable and vituperative behavior, its pseudoaddisonian symptoms, and the color of the urine are described. A case illustrating intermittent porphyria and many of the factors discussed is presented in considerable detail. The author suggests that this disease may be far more widespread than is currently believed.

Despite all extravagant claims, no specific treatment has been found for intermittent porphyria. Severe abdominal cramps can be controlled with Demerol or tetraethylammonium chloride, which will also relieve neuropathic pains. Some benefits may accrue from the use of Prostigmin and mephenesin, from sympathectomy, massive doses of vitamins, liver preparations, or adrenocortical hormones. The use of corticotropin and the adrenal steroids is justified in treating fulminating acute porphyria, which is so often fatal and which is reportedly relieved by these agents. Chloral hydrate and paraldehyde are preferred for sedation, and the barbiturates are avoided. The effects of Rauwolfia serpentina are under study. Although porphyria is difficult to manage, unnecessary surgery can be avoided, drug therapy limited, and morale and physical fitness maintained. Cutaneohepatic (mixed) porphyria, the symptoms of which sometimes suggest intermittent porphyria, is discussed as are the miscellaneous porphyrias and the toxic porphyrinopathies.

The need for screening the urine for porphobilinogen, uroporphyrin, and coproporphyrin whenever there are obscure abdominal colic, hysterical symptoms, and neuropathies is once again stressed, and rough screening tests for measuring abnormal amounts of these substances are described.

MANAGEMENT OF THE DILATED URETER

Calling for more adequate urologic, neurologic, and radiologic evaluation of the dilated ureter, for logical surgical therapy, for strict attention to bacterial flora, and for follow-up throughout the period of growth, Donald A. Charnock discusses the child with a urologic handicap in *The Journal of the American Medical Association,* February 12, 1955.

Ureteral dilatation in children results from mechanical obstruction or neuromuscular dysfunction, or occurs sometimes even when no cause is apparent. Dilatation caused by a congenital or acquired obstruction distal to the ureter is properly described as hydroureter. The lumen of the ureter is widened and elongated by hydrostatic pressure, which eventually causes angulation and convolution of the ureter. The ureteral orifices are incompetent. When the loops become adherent, secondary obstruction arises, further slowing the flow of urine from the kidney to the bladder. Hydronephrosis and bladder dilatation are associated conditions, and infection is a constant threat.

The purpose of treatment is to relieve the obstruction and to correct ureteral deformities. Suprapubic drainage may relieve early dilatation and reduce hydronephrosis. More advanced or more extensive ureteral angularity is corrected by plastic operation. Segmental ureteral resection is successful in the young child. Meatotomy will relieve deformities of the ureterovesical orifices. Charnock describes his technic for correcting hydroureter, his staging of the procedure, and also reviews problems complicating the treatment of megaloureter resulting from faulty innervation or from some little understood cause.

THE IMPORTANCE OF GLAUCOMA WITH INCREASING AGE

Early diagnosis of glaucoma, which causes 7 to 15 per cent of the nation's blindness, becomes increasingly important as our aging population grows. With 11.5 million people older than 64, a review of a disease that has a predilection for the older person is particularly timely. Conrad Berens, M.D., and Arnold S. Breakey, M.D., discuss glaucoma in the *Journal of The American Geriatrics Society,* March, 1955.

Changes in intraocular tension result from an increase in inflow or a decrease in outflow of the fluids that nourish the eyeball. Reduced corneal and scleral elasticity interfere with ocular adjustment to such changes which, in turn, affect the size of the choroidal vessels and the vessels of the ciliary body. When the eye fails to adjust to increased internal tension, glaucoma follows. The disease can be arrested by treatment, but the blindness that it causes is irreversible.

Primary glaucoma may be acute uncompensated, chronic uncompensated, compensated, or absolute. The first is common in the forties and later, appears suddenly, causes intense pain, lacrimation, conjunctival congestion, and reduced vision, but often goes unrecognized because the accompanying headaches, prostration, nausea, vomiting, and fever suggest indigestion. The differential factors that point to glaucoma are suddenness of onset, depth of congestion, cloudiness of the cornea, the oval shape and dilated state of the pupil, seriously reduced vision, severe pain along the fifth cranial nerve, the prostration and the vomiting, and increased intraocular tension. In chronic uncompensated glaucoma, the symptoms are milder, and diagnosis cannot depend on a measure of the intraocular tension,

but rather on the glaucomatous changes which careful study of the visual fields will show. Superficial examination might suggest cataract, and proper treatment might be delayed because of the incorrect diagnosis.

In compensated glaucoma, the pupil is rigid and dilates slowly, the anterior chamber is shallow, visual acuity is diminished, and changes can be seen in the lens and the optic disk. Intraocular pressure varies and is increased; changes in refraction and dark adaptation are characteristic, and defects can be seen in the visual fields. Signs of hyperemia or inflammation and abnormalities of the conjunctiva, cornea, or sclera need not be apparent.

Since any of these types of glaucoma, unless treated in time, will lead to absolute glaucoma and blindness caused by atrophy and retinal degeneration, it becomes increasingly important for the physician to be familiar with the signs and symptoms of the disease and to be able to measure intraocular tension as part of the routine physical examination of older persons. The author discusses diagnosis, prognosis, and treatment and describes an ocular hypertension indicator that permits early recognition of the disease by a physician.

POSTURAL SHOCK IN PREGNANCY

Ralph L. Wilkening, M.D., and others discuss the shock that is sometimes experienced late in pregnancy by a patient in dorsal recumbency. They present the histories of four patients experiencing it, who were studied and treated at Kern General Hospital, and report their observations in *California Medicine,* March, 1955.

Signs and symptoms of shock were noted in two patients during early labor and in two others during routine prepartum examinations. The faintness, nausea, shortness of breath, pallor, perspiration, and pronounced hypotension, and tachycardia were relieved as soon as the patient changed her position. The symptoms arise from compression of the inferior vena cava by the near-term, flaccid uterus when the patient lies on her back. The blood pools in the legs, the return flow to the heart and lungs is reduced, and the cardiac output is diminished. The phenomenon is not more widespread because of the variance in relationship of the vena cava to surrounding structures.

Awareness of this syndrome might spare the patient from unnecessary treatment for shock. Hypotension from other causes can be aggravated by posture, and borderline hypotension due to posture may precipitate vascular collapse if, in such circumstances, some other agent further lowers the blood pressure. Before shock is treated in late pregnancy, it would be judicious to re-examine the patient while she is lying on her side. Whatever the cause of shock during late pregnancy, a change of posture might alleviate it.

A COMPARISON OF VAGOTOMY AND GASTRIC RESECTION FOR GASTROJEJUNAL ULCERATION: A FOLLOW-UP STUDY OF 310 CASES

A study based on Mayo Clinic records on 617 operations performed on 586 patients for gastrojejunal ulcers that occurred after gastroenterostomy or after gastric resection and comparing the results obtained by vagotomy and by gastric resection is presented by Waltman Walters, M.D., and others in *Surgery, Gynecology and Obstetrics,* January, 1955.

In all, 143 operations consisted of vagotomy alone or combined with other procedures; the rest were, for the most part, gastric resections. All 143 patients who had vagotomy and 158 —approximately every third patient of those who had gastric resection—were included in the comparative study. The 301 were further subdivided: in 115 the gastrojejunal ulcer had occurred after gastric resection, and in 186 after gastroenterostomy. Such subgrouping was prompted by clinical differences between peptic ulcers that develop after gastric resection and those that follow gastroenterostomy; treatment of the two types also varies.

Significant preoperative clinical features were compared with the same features postoperatively. The etiology and incidence of gastrojejunal ulcer, its prevalence by age and sex, histories of earlier peptic ulcer operations, and the complications are discussed in terms of this series. Diagnostic aids used, treatment given, and death rates after gastric resection and after gastroenterostomy are compared. Finally, treatment results in both groups are analyzed on the basis of the treatment selected. Results of surgical treatment for gastrojejunocolic fistula, so often fatal in the past, are tabulated for the twenty patients in this series who were operated on and lived.

Return visits or questionnaires formed the basis of a 1-8 year follow-up of 242 of the 301 patients. Vagotomy, alone or combined, was considered the best treatment for gastrojejunal ulcers arising after gastric resection: results were excellent in 70.5 per cent of the 78 patients treated by vagotomy alone, in all of the nine patients treated by vagotomy and gastric re-resection, and in 57.1 per cent of the seven traceable patients treated by gastric re-resection alone.

Preferred treatment for gastrojejunal ulcer after an earlier gastroenterostomy is to take down the anastomosis, excise the ulcer, and do adequate gastric resection. The results were excellent in 86.5 per cent of the traceable patients so treated, as against 77.8 per cent among those treated by vagotomy.

Gastrointestinal hemorrhages recurred in 10.4 per cent of those who had vagotomy, as against 3.4 per cent of those who had gastric re-resection.

The mass of detailed data suggested that gastric resection excelled vagotomy for treating gastrojejunal ulcer after gastroenterostomy, but that vagotomy played an important role in the management of gastrojejunal ulcer after partial gastric resection. The approach used in vagotomy—whether transabdominal or transthoracic—had no significant bearing on the final results.

POTENTIATION OF HYPNOTICS AND ANALGESICS

Robert Wallis, M.D., discusses his experience with the use of chlorpromazine (a new phenothiazine derivative) to supplement sedation which alone had failed to relieve insomnia in 17 patients. His results were reported in the *New York State Journal of Medicine,* January 15, 1955.

The patients, despite treatment with barbiturates and other soporifics, had continued to have difficulty sleeping. When chlorpromazine was added, however, all of them slept 8-10 hours and awakened refreshed, regardless of the cause of the insomnia. Chlorpromazine depresses the hypothalamic centers of the sympathetic nervous system, thereby inhibiting cenesthesia. It appears to potentiate and augment the action of hypnotic, narcotic, and analgesic preparations, often making it possible even to lower the dosage of narcotics. With 25-50 mg. chlorpromazine, as little as ¼ to ½ grain of phenobarbital and 12 grains of chloral hydrate were adequate. The effects of ingesting alcohol before or during chlorpromazine therapy were undesirable. It failed to relieve migraine, premenstrual pelvic pain, or radicular sciatica with sacroiliac dislocation, but relieved the pain and induced an emotional indifference toward impending death in a patient with terminal cancer.

MOBILIZATION OF THE STAPES TO RESTORE HEARING IN OTOSCLEROSIS

An earlier report, given in May, 1953, on restoring hearing by mobilizing the stapes in otosclerosis is followed up by Samuel Rosen in the *New York State Journal of Medicine,* January 1, 1955.

Deafness that has been considered irreversible is caused by ankylosis of the footplate of the stapes in otosclerosis. Sound fails to reach the cochlea. However, it is possible to free and

mobilize the fixed stapedial footplate by manipulation through the external ear, thereby restoring hearing. The technic of mobilization is fully described, and the instruments needed in performing the operation are illustrated. This operation was performed on five persons, whose case histories are given. In all five cases, the mobilizing technic improved hearing, and in two of them hearing was restored to normal. Follow-up on these cases has continued for from 16 to 25 months. The results show that reankylosis of the stapedial footplates has not taken place, and that the audiologic improvement has been maintained. Should ankylosis recur, the stapes could once again be mobilized and, should this maneuver fail, the possibility of doing a fenestration operation to create a new window into the labyrinth to permit sound to reach the cochlea would still remain.

No more than a day's hospitalization is required for the mobilization. No postoperative vertigo is experienced, and no special postoperative treatment is necessary. The author does, however, point out that it takes fifty practice operations on a cadaver to acquire the skill required to mobilize the stapes successfully in a living patient.

MILK-ALKALI SYNDROME

Donald A. Scholz, M.D., and F. Raymond Keating, Jr., M.D., stress the hazards and importance of the milk-alkali syndrome on the basis of clinical and laboratory observations on eight patients studied at the Mayo Clinic and report their observations in the *A.M.A. Archives of Internal Medicine*, March, 1955.

Four of the patients had had ulcer symptoms for 20 years or longer; the other four had had them for 2 to 7 years. Their daily intake of milk ranged from 1 glass to 3 quarts. One patient drank a pint of half milk, half cream, in addition to a quart of milk a day. Each patient took some form of absorbable alkali—alkaline or Sippy powders, sodium bicarbonate, or a proprietary antacid—in amounts ranging from 4 to 6 tablets or teaspoonsful to as much as 2 to 2½ lb. a week. Serum levels of calcium ranged from 11.2 to 15.6 mg. per 100 cc. and of inorganic phosphorus from 2.6 to 6.2 mg. per 100 cc. Blood urea values ranged from 85.4 to 234 mg. per 100 cc., and the CO_2-combining power ranged from 22 to 41.2 mEq. per liter. It took from 4 to 16 days for blood calcium levels to return to normal after ulcer diets low in calcium content were introduced.

The syndrome is seen in ulcer patients after prolonged treatment with milk and absorbable alkalis. It is recognizable by hypercalcemia that manifests itself without hypercalciuria or hypophosphatemia, by renal insufficiency and azotemia, by alkalosis, and by ocular lesions that resemble band keratitis. The symptoms usually disappear when milk and absorbable alkalis are omitted from the diet.

The authors present a case to illustrate some of the difficulties encountered in differential diagnosis, in addition to the histories of their 8 cases. Successful differentiation from primary hyperparathyroidism might obviate unnecessary and useless surgical exploration, and the recognition of a renal lesion arising from duodenal ulcer therapy might reduce serious surgical risks.

FACTORS DETERMINING MORTALITY IN PATIENTS WITH ACUTE HEAD INJURY

A new classification that approaches acute head injury both clinically and pathologically and permits a more objective and complete view of the patient is based on experience with 1,000 consecutive patients treated for head injuries at Hahnemann Hospital, Philadelphia. Alex W. Ulin, M.D., and his associates discuss the problem in *The Journal of the American Medical Association*, February 5, 1955.

Death after acute head injury may result from brain damage, shock, asphyxia, simultaneous injury to some other system, or coincidental disease. Other potential causes may be infection, radiation damage, and mass catastrophe. Although the Hahnemann over-all death rate of 7.4 per cent for the series is excellent, critical analysis proved that from 40 to 60 per cent of those who had severe head injuries and who were treated surgically died. Since severe injuries comprise from 10 to 15 per cent of all acute head injuries, the outlook for the severely injured remains poor. To lower death rates in this category, efforts to relieve cerebral edema by subtemporal decompression and decompressive craniectomies have been abandoned at Hahnemann where the staff has tended also to rely less on repeated spinal taps and drainage and to avoid regimens evoking serious dehydration and using intravenous administration of hypertonic solutions. Instead, more conservative measures, designed to support the patient as a whole, have been followed, and the indications for surgery of any kind have been closely observed. The Hahnemann classification emphasizes watching for shock, paradoxical respiration, ruptures of vital organs, spinal cord injuries, and other important damage which might otherwise be overlooked in treating a skull injury. Roentgen diagnosis, particularly in chest and abdominal injuries, is considered essential whether the patient is in shock or not. Antibiotics are used routinely on patients bleeding or losing cerebrospinal fluid through the orifices of the head.

Coma is an extremely important factor in assessing the seriousness of cerebral damage; in this series, it was the sole manifestation of brain injury among 40 per cent of the patients. The indications for exploratory burr holes are persistent, deepening, or intermittent coma; progressive deterioration, regardless of the rate; persistent or progressive abnormal neurologic signs; prolonged stationary course; and a critical condition when an accurate history and clinical details are lacking. The most serious complication of head injury, respiratory embarrassment, is treated by endotracheal suction or tracheostomy. Airways are cleansed and laryngeal obstructions relieved by tracheostomy in patients with both head and chest injuries. Tracheostomy is helpful also when the comatose state exceeds 24 hours.

ENURESIS

A Critical Review of the Symptoms

Enuresis is a symptom for which many kinds of treatment, mostly unscientific, have been tried. Symptoms and suggestions for therapy are presented by Joseph M. Teicher, M.D., in the May, 1955, issue of *California Medicine*.

The establishment of bladder control at the appropriate age is important because it means self-control, which is one of the foundations of education, that of enabling the child to be consistent in his efforts to attain certain goals, even if that requires labor, time, and patience. Children who wet the bed are often poor students, however intelligent, for they cannot apply themselves, concentrate, or obey orders. They want to succeed immediately or they give up. They frequently get into trouble sexually. On the other hand some enuretic children are dominated by their mothers and are timid and lacking in initiative. This type is often conspicuous by his unkempt, dirty appearance and by his whining attitudes and his inability to defend himself against other children.

There has been no convincing demonstration of organic disease in the urinary system of the great majority of bedwetters. A relatively large proportion of such children have abnormal encephalograms, but, as the author points out, coincidence does not always spell causal relationship. The same is true of phimosis, spina bifida, local irritation, balanitis and the like when they are found.

The influence of training and parental attitudes are much more frequent and important factors. Overprotection and maintenance of the infantile state are causes, as is lack of opportunity for training in rural homes and in crowded urban areas.

Therapeutic approaches, in the absence of organic disease, are based on discontinuance of punishment and disparagement and avoidance of drugs and manipulations. Helping parents in their attitudes and helping and encouraging the child "who is to be considered an individual and not merely a bladder" is likely to succeed.

Book Notices

CLINICAL DIAGNOSIS. By Elmer G. Wakefield, B.S.A., BSc., M.D., F.A.C.P., Diplomate of the American Board of Internal Medicine; Consulting Physician, Section of Medicine, Mayo Clinic; and Associate Professor of Medicine, Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota, Rochester, Minnesota. Cloth. Pp. 1611, with illustrations. Price $22.50. Appleton-Century-Crofts, 35 W. 32nd St., New York 1, 1955.

This single volume comprehensively covers the nonspecialty methods and technics by which any physician can correctly diagnose the large percentage of cases which present themselves to him.

For convenience of reference, the book is divided into three main sections with 531 pages devoted to the diagnosis of regional diseases, 660 pages to systemic diseases, and 354 pages to diseases which affect the body as a whole.

The author is a well-known diagnostician on the staff of the Mayo Clinic and has, of course, supplemented his own experience with that of associates at Mayo. The detailed case histories of the Clinic have been available to him, and the entire manuscript was read and edited by the Clinic's editors before being presented for publication.

The result is a comprehensive and authoritative work of more than 1,600 pages devoted exclusively to practical methods and tested technics. According to the author:

> In order to use this book it is necessary to ascertain the topographic area or the system wherein the manifestations of spurious or organic disease arise. Thereafter, in about seven out of ten cases, diagnosis can be made by the physician who employs his own senses, under the control of his own knowledge and experience, aided if necessary by what this book contains. In the remaining three cases, diagnosis depends on endoscopy, roentgenology or laboratory determinations, as these are evaluated by the internist, surgeon or other specialist.

MAXILLOFACIAL ANATOMY With Practical Applications. By Harry H. Shapiro, D.M.D., Assistant Professor of Anatomy, College of Physicians and Surgeons, Columbia University; Visiting Lecturer in Applied Anatomy, Tufts College; Consultant, U.S. Army Medical Service Graduate School, Walter Reed Hospital, Washington, D.C.; Attending Maxillofacial Surgeon in Plastic Surgery, Manhattan Eye, Ear, and Throat Hospital, New York City; Consultant, Cornwall Hospital, Cornwall, New York. Cloth. Pp. 392, with illustrations. Price $12.00. J. B. Lippincott Company, West Washington Square, Philadelphia 5, 1954.

This book is a detailed, correlated study of the principles of structure and movement of the anatomic components of the maxillofacial area and a practical application of these principles. Anatomy is taught in relation to its usefulness in practice, with the aim of heightening interest in and promoting better understanding of the subject matter. Significant applications of the principles are incorporated into the text. In addition to normal structure, various abnormal and anomalous anatomic conditions are discussed in relation to treatment.

The text is specifically intended for students and practitioners of dentistry and medicine and for others desiring practical information on the morphologic, functional, and roentgenologic anatomy of the maxillofacial region and associated structures of the head and neck.

PULMONARY DISEASES. Edited by Roscoe L. Pullen, A.B., M.D., F.A.C.P., Professor of Medicine and Dean, University of Missouri School of Medicine, Columbia, Missouri; Consultant to the Surgeon General, Department of the Army, Washington, D. C. Cloth. Pp. 669, with illustrations. Price $15.00. Lea & Febiger, Washington Square, Philadelphia 6, 1955.

Mass chest x-rays and intracutaneous testing and the resultant discovery of active pulmonary disease and asymptomatic or incipient chest conditions have caused a renewal of interest in pulmonary diseases. In addition, increased knowledge of the physiology of respiration has resulted in the development of diagnostic technics and therapeutic measures that have improved the clinical management of many lung diseases. This book considers the field of pulmonary diseases from the clinical viewpoint. Where it was thought necessary to aid clinical understanding, brief reviews of the anatomy, physiology, and pathology of various processes have been included. Emphasis, however, is mainly on diagnosis and treatment.

Nineteen other authors, all leaders in their fields, have collaborated with Dr. Pullen in writing this book. Some of the subjects considered are bronchitis, bronchial obstruction, bronchiectasis, bronchial asthma, benign lung tumors, bronchiogenic carcinoma, pulmonary infections and pneumonias, pulmonary mycoses, animal parasitic infections of the lungs, and structural alterations of the lungs.

The volume is intended as an authoritative guide for general practitioners, chest specialists, surgeons, and students.

ESSENTIALS OF MEDICINE. The Art and Science of Medical Nursing. By Charles Phillips Emerson, Jr., A.B., M.D., Associate Professor of Medicine, Boston University School of Medicine; Member, Robert Dawson Evans Memorial Laboratory; Visiting Physician and Director of Clinical Laboratories, Hematology Clinic and Radioisotope Division, Massachusetts Memorial Hospitals; Consultant in Hematology, U.S. Public Health Service Hospital, Brighton, and Attending Physician, Veterans Administration Hospital, Boston; and Jane Sherburn Bragdon, R.N., B.S., Associate Director, School of Nursing, Massachusetts Memorial Hospitals, and Clinical Assistant in Medical and Surgical Nursing, Boston University School of Nursing. Ed. 17. Cloth. Pp. 922, with illustrations. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1955.

This text, which has been in existence for nearly 50 years, is now in its seventeenth edition. It has been extensively revised, rewritten, and rearranged, and many important additions have been made. The scope has been extended by adding a unit on nursing in disaster. In this section consideration is given to various specific problems which may be anticipated in the event of a major catastrophe, whether it is of military or natural origin.

Increased emphasis is given to nursing care, and nurse-patient relationships are stressed. At the end of every unit situations are described and questions asked for the purpose of stimulating classroom or ward discussion. The role of the nurse in diagnostic tests and procedures, in geriatric care, and in disasters, is considered. The socioeconomic and psychologic factors in patient care are integrated throughout, and modern concepts of tuberculosis nursing are included.

RECENT DEVELOPMENTS IN PSYCHOSOMATIC MEDICINE. Edited by Eric D. Wittkower, M.D., Associate Professor of Psychiatry, McGill University, and R. A. Cleghorn, M.D., D.Sc., Associate Professor of psychiatry, McGill University. Cloth. Pp. 495. Price $10.00. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1954.

A reference compiled by twenty-nine contributors who have made significant contributions to research and clinical investigation in psychosomatic medicine, this book's purpose is to describe modern developments in psychosomatic medicine. Some of the questions which the volume attempts to answer are, "What do we now know? What are the theoretical links which bridge the islands of our certain knowledge? What suggestions loom from uncertain seas spanned by our theories?

In what direction must we now venture in order to expand, consolidate and integrate our present understanding?" With the hope of making stimulating argument, the editors encouraged each contributor to express his views freely and not to restrain any tendency to be provocative.

Physiologic and somatic pathology are correlated with the psychologic disturbance. The text aids in establishing the etiologic picture and dynamics of the particular disorder and in choosing the therapeutic procedures which are most apt to lead to the quickest and best recovery.

The book is divided into two sections: The first contains general articles surveying and evaluating current research. The other section, by far the greater part of the work, is specific and clinical. Some of the topics discussed are the interplay between endocrine and psychologic dysfunction; the thyroid; psychologic aspects of diabetes mellitus, of gynecology and obstetrics, of skin diseases, of patients with peptic ulcer, of nonspecific ulcerative colitis, and of pulmonary tuberculosis; rheumatoid arthritis; cardiovascular disorders, heart disease, and hypertension; venereal disease; psychosomatic medicine and pediatrics; disablement; and psychosocial medicine.

According to the editors, the book is intended for anyone genuinely interested in psychosomatic medicine—those whose interest is in patients rather than in diseases.

LEGG-CALVE-PERTHES SYNDROME and Related Osteochondroses of Youth. By Charles Weer Goff, M.D., Assistant Clinical Professor of Orthopaedic Surgery, Yale University School of Medicine; Attending Orthopaedic Surgeon, Newington Home and Hospital for Crippled Children, St. Francis Hospital, Hartford; Visiting Professor of Physical Anthropology, Hartford Seminary Foundation; Member, Society for Research and Child Development. Cloth. Pp. 332, with illustrations. Price $10.75. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

In this monograph the author sets out to answer certain questions pertaining to Legg-Calvé-Perthes disease, and he has done a great deal of research on the subject in order to provide the answers. Some of the questions are, "What kind of children develop these disturbances?" "Who are their parents, and does heredity play a part in the development of the disorder?" "In children of what races does it develop?" "What effect do the restraints used have on the personality of the child?"

The historic aspects of the syndrome are painstakingly presented. The physiologic, psychologic, and sociologic views of the subject are discussed fully. The various etiologic factors are considered. Treatment is critically discussed. Other osteochondroses are also considered.

It is unfortunate that such a thorough study shows a lack of editorial attention.

FLUID AND ELECTROLYTES IN PRACTICE. By Harry Statland, M.D., Associate in Medicine, University of Kansas School of Medicine; Consultant in Medicine, Veterans Administration Hospital, Kansas City, Missouri; Attending Physician, Menorah Medical Center, Kansas City, Missouri. Cloth. Pp. 206, with illustrations. Price $5.00. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1954.

Requests of many students to put his lecture material in permanent form caused Dr. Statland to expand his classroom notes and assemble them in this volume.

The book is in two parts: In the first are presented general principles of fluid movements, and major volume, concentration, and acid-base balance abnormalities. Management of the surgical patient is particularly stressed in this section. In the second section, management of fluid balance is considered in relation to special conditions: heart disease; kidney and urologic diseases; diabetic acidosis; burns; cirrhotic ascites; and toxemias of pregnancy, and in pediatrics.

The author apologizes for possible oversimplification of the material, stating that his intention was to keep the text intelligible to those practicing physicians who have long been removed from the basic sciences. Instead of being a cause for apology, this reviewer feels that its simplicity is an outstanding feature of the text. In addition, there are numerous explanatory tables and illustrations which aid in clarification of important concepts.

TRANSFERENCE: Its Meaning and Function in Psychoanalytic Therapy. By Benjamin Wolstein, Ph.D., Clinical Psychologist, Low Cost Psychoanalytic Service, W. A. White Institute of Psychiatry, Psychoanalysis and Psychology; Psychologist, Therapeutic Services, Postgraduate Center for Psychotherapy, New York. Cloth. Pp. 206. Price $5.00. Grune & Stratton, 381 Fourth Avenue, New York 16, 1954.

This is a technical book for the dynamic psychiatrist who is engaged in the regular practice of psychotherapy. It is in line with contemporary psychoanalysis and its discussions are in the terms of modern theory. Much of the work relates to a comparison of Freud's earlier teaching and the apparent influence of hypnosis on his handling of the transference problem. Freud, who was not a hypnotist, gives much evidence in his writings of the impact of his contact with hypnosis. It is only in recent years—when the therapist's role in the transference was recognized—that this became evident.

The book shows clearly the place of the teachings of Harry Stack Sullivan on modern psychiatric therapy and practice. The place of the therapist is implied by the author: "What the analyst says or does is as important in the formation of transference distortions as the potential for typical sorts of distortion which the patient brings to analysis."

For the psychiatrist and psychiatric student the book has much of interest, but physicians interested in the handling of psychosomatic conditions will find much information which will help in their therapy.

THOMAS J. MEYERS, Ph.D., D.O., F.A.C.N.

ULTRASONIC AND ULTRASHORT WAVES IN MEDICINE. By Johanna M. Van Went, M.D., Director Institute for Physical Medicine and Rheumatism, Amsterdam. Cloth. Pp. 384. Price $9.00. The Elsevier Press, 402 Lovett Boulevard, Houston, Tex., 1954.

In this monograph a relatively new branch of physical medicine, ultrasonic and ultrashort waves, is reviewed. Biologic investigation during the past 10 years in Europe and in this country is included. The use of ultrasonic waves in diseases of the spine, joints, chest, abdomen, ear, and skin are discussed. Ultrashort waves are considered in disorders of the central and peripheral nervous system, and the heart and vessels, in nonrheumatic joint disorders, muscular affections and rheumatism, and in gastrointestinal, respiratory, and urogenital diseases. There are also sections on hyperthermia, irradiation of the hypophysis, and microwaves.

THE HEPATIC CIRCULATION AND PORTAL HYPERTENSION. By Charles G. Child, III, M.D., Professor of Surgery, Tufts College Medical School; Chairman, Department of Surgery, New England Center Hospital; Formerly Associate Professor of Clinical Surgery, Cornell University Medical College, Attending Surgeon, New York Hospital. From the Department of Surgery and the Laboratory of Surgical Research of the New York Hospital—Cornell Medical Center. Cloth. Pp. 444, with illustrations. Price $12.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1954.

Current interest in the relationship between liver disease and the hepatic circulatory systems has resulted in investigations of portal hypertension, the existence of which has been suspected for a long time but which has only recently been accurately defined. This renewal of interest and the problems

which have arisen because of the discrepancies resulting from various investigations have stimulated the preparation of this volume.

The monograph reviews the historical background of liver diseases and the present knowledge of the comparative anatomy of hepatic circulation, the embryology of the liver and its blood vessels, anomalies of the hepatic vasculature, gross and microscopic anatomy and normal physiology of the intrahepatic vasculature, and intrahepatic changes in liver disease.

In his discussion of portal hypertension the author has analyzed the case reports of the 362 venovenous shunts performed to date for portal hypertension.

Pancreaticoduodenectomy with resection of the portal vein is discussed, as is the preoperative and postoperative water, electrolyte, and protein balance in patients with cirrhosis.

In the appendices Dr. Child describes his experiments with Macaca mulatta monkeys and with dogs, and abstracts the reports of all the venovenous shunts which he has performed on patients.

ANATOMY FOR SURGEONS: Volume 1. The Head and Neck. By W. Henry Hollinshead, Ph.D., Professor of Anatomy, Mayo Foundation, University of Minnesota; Head of the Section of Anatomy, Mayo Clinic, Rochester, Minnesota. Cloth. Pp. 560, with illustrations. Price $12.00. Paul B. Hoeber, Medical Book Department of Harper & Brothers, 49 E. 33rd St., New York City 16, 1954.

Rather than presenting a complete descriptive anatomy, this book considers the regional anatomy of particular importance to the surgeon and interprets anatomic facts and concepts which have been found useful to the surgeon. Dr. Hollinshead taught anatomy to medical students for many years and at present is consultant in anatomy to the physicians of the Mayo Clinic and Foundation. In addition to the author, one or more surgeons in active practice read and criticized each chapter for its content and applicability to surgery.

"The Head and Neck" is the first of three volumes of a work which will also include "The Thorax, Abdomen, and Pelvis" and "The Back and Limbs."

Indications for and details of specific operations are not included because the author rightfully feels that they belong to a text on surgery rather than one on anatomy. However, certain anatomic facts and concepts which are of importance only as they pertain to surgery are considered. Illustrations are diagrammatic and easily comprehended.

The volume is intended to review broad basic anatomy for the younger surgeon; to discuss the details which are the bases for the work of the more mature surgeon; and, for the latter also, to provide a refresher for details which may be outside his special field and to serve as a ready reference for details in his own specialty which he cannot keep constantly in mind.

ATLAS OF ORTHOPEDIC TRACTION PROCEDURES. By Carlo Scuderi, B.S., M.D., M.S., Ph.D., Clinical Associate Professor of Surgery, University of Illinois; Professor of Surgery, Cook County Graduate School; Attending Surgeon, Cook County Hospital; Chairman of Department of Orthopedic Surgery, St. Elizabeth's Hospital and Columbus Hospital; Senior Orthopedic Surgeon, Alexian Brothers' and St. Anne's Hospitals; Consulting Orthopedic Surgeon, Augustana Hospital, Chicago; Consulting Orthopedic Surgeon, McNeal Memorial Hospital, Berwyn, Illinois; Member of American Academy of Orthopedic Surgery, Clinical Orthopedic Society, American Association for the Surgery of Trauma, Western Surgical Association, American Medical Association, American College of Surgeons. Cloth. Pp. 230, with illustrations. Price $12.50. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1954.

The author's purpose in preparing this book was "to fill a definite gap in orthopedic literature by photographs, line drawings and simple descriptive language." The illustrations are clear and understandable; on the page opposite the illustration there is a concise description of the device and principles involved, explaining simply but completely how to set up orthopedic traction. The book should be helpful to all working with orthopedic patients.

THE PATHOLOGY OF TRAUMA. By Alan Richards Moritz, M.D., Professor of Pathology and Director of the Institute of Pathology of the School of Medicine of Western Reserve University, Cleveland, Ohio. Ed. 2. Cloth. Pp. 414, with illustrations. Price $8.50. Lea & Febiger, Washington Square, Philadelphia 6, 1954.

Because any book dealing with applied science rapidly becomes obsolete and because so much was learned about violent injury from recent wars, a new edition of this book became necessary. Data from industry, transportation, war, and crime have been correlated. New interpretations of the physiologic changes due to trauma are presented, and psychosomatic complications of trauma are also considered.

The leading causes of violent injury are surveyed, and the manner in which they produce functional and structural disturbances, their pathologic characteristics, pathogenesis, sequelae, and medicolegal aspects are discussed.

The subjects covered include mechanical injuries; trauma and infection; trauma and tumors; mechanical injuries of the cardiovascular, respiratory, alimentary, biliary, urogenital, central nervous, and musculoskeletal systems; and medicolegal autopsy, the last being a new chapter for this edition.

The book is recommended for clinicians, surgeons, medical examiners, coroners, industrial physicians, and lawyers.

MODERN DIAGNOSIS AND TREATMENT OF THE MINOR VENEREAL DISEASES. By Orlando Canizares, M.D., F.A.C.P., Associate Professor, Clinical Dermatology and Syphilology, New York University, Post-Graduate Medical School; Consultant, Veterans Administration Hospital, Bronx, New York; United States Public Health Service Hospital, Staten Island, New York. Cloth. Pp. 121, with illustrations. Price $3.75. Charles C Thomas, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

This monograph is timely in that it serves as a reminder that venereal disease is more than gonorrhea and syphilis, as the average physician in practice has very likely come to think. Antibiotics are considered as a panacea so that diagnosis is no longer needed. Reading this text brings to mind the fact that chancroid, granuloma inguinale, and lymphogranuloma venereum are still with us. The coverage of these diseases is most comprehensive; each is discussed in all its aspects. Probably of the greatest value is the table reviewing the summary of laboratory diagnosis for each disease, which gives the pertinent information at a glance. Antibiotic therapy with the dosage schedules is presented in detail.

The book should be available for reference, particularly in hospitals and clinics.

A. P. Ulbrich, D.O.
D. Koprince, D.O.

HUMAN PHYSIOLOGY. By Bernardo A. Houssay, M.D., Professor of Physiology; Director of the Institute of Biology and Experimental Medicine, Buenos Aires, Argentina; Juan T. Lewis, M.D., Professor of Physiology; Director of the Institute for Medical Research, Rosario, Argentina; Oscar Orías, M.D., Professor of Physiology; Director of the Mercedes and Martin Ferreyra Institute for Medical Research, Córdoba, Argentina; Eduardo Braun-Menéndez, M.D., Professor of Physiology; Member of the Institute of Biology and Experimental Medicine, Buenos Aires, Argentina; Enrique Hug, M.D., Professor of Pharmacology of the School of Medicine, Rosario, Argentina; Virgilio G. Foglia, M.D., Professor of Physiology; Member of the Institute of Biology and Experimental Medicine, Buenos Aires, Argentina; Luis F. Leloir, M.D., Director of the Institute for Biochemical Research, Campo-

mar Foundation, Buenos Aires, Argentina. Translated by Juan T. Lewis, M.D., and Olive T. Lewis. Ed. 2. Cloth. Pp. 1177, with illustrations. Price $12.00. McGraw-Hill Book Company, 330 West 42nd St., New York 36, 1955.

Dr. Houssay, winner of the Nobel prize for Medicine in 1947, and his associates continue in this, the second, edition of their text their policy of presenting the various aspects of physiology in their chemical and physical relationships. Certain indispensable information is omitted because, if it is not known, it can easily be found in texts of anatomy, histology, embryology, biochemistry, and biophysics. Other subjects of great importance are treated only briefly because they will be taken up in detail in specialized medical studies. Secondary, debatable, and material of clinical interest is included in small type. The authors have refrained, as much as possible, from reporting their own work more fully than that of others.

The latest data in the field of physiology have been included in this revision. There have been extensive modifications and additions to the sections on nutrition, carbohydrate metabolism, the endocrine glands, sexual physiology, and the electrical activity of the heart. Recent advances in the mechanisms of conduction of the nerve impulse and synaptic transmission have also been incorporated.

The text is recommended for medical students.

CASIMIR FUNK, Pioneer in Vitamins and Hormones. By Benjamin Harrow. Cloth. Pp. 209, with illustrations. Price $4.00. Dodd, Mead & Company, 449 Fourth Ave., New York, 1955.

This biography presents a full-length word portrait of Casimir Funk, the man who first isolated the antiberiberi substance, for which he coined the term "vitamine," now spelled without the final "e," although the original was taken from "amine," which Funk wrongly believed the substance to be.

In 1912 he projected his hypothesis which "laid the very foundation stone for the entire subject of vitamins." He proposed that there were at least four vitamins. His conclusions that such diseases as beriberi, pellagra, rickets, and scurvy were due to deficiency of specific chemical substances marked a milestone in medical science. A book on vitamins by Funk was translated into many languages.

Funk came to the United States shortly after the outbreak of World War I. With a collaborator he achieved a concentrated fraction of vitamins A and D, later marketed as Oscodal, the first vitamin product accepted by the American Medical Association. Over 150 scientific papers by Funk and collaborators have been published throughout the world.

NONTUBERCULOUS DISEASES OF THE CHEST. Sponsored by the American College of Chest Physicians. Edited by Andrew L. Banyai, M.D. Cloth. Pp. 1139, with illustrations. Price $18.75. Charles C Thomas, Publisher, 301-327 East Lawrence Ave., Springfield, Ill., 1954.

The mortality and morbidity of chest diseases, nontuberculous and tuberculous, are tremendous. The American College of Physicians has sponsored this volume on nontuberculous diseases of the chest as a companion to one on pulmonary tuberculosis which it sponsored earlier. In filling the need for a comprehensive informative guide to better understanding and more efficient treatment of nontuberculous chest diseases, the thirty-seven contributors from this country, Argentina, England, India, and Mexico have drawn upon their vast clinical experience. The text opens with a discussion of the physiology and pathologic physiology of respiration.

Because of modern transportation and long-distance travel, diseases peculiar to or endemic in one geographic location may appear elsewhere. With this in mind, available diagnostic procedures are critically reviewed, and new methods of managing various clinical entities are offered.

In addition to the more common chest diseases, such as the pneumonias, bronchial asthma, carcinoma, and atelectasis, congenital chest diseases are discussed, as are pathologic conditions resulting from metabolic disturbances, hemopoietic system diseases, lymphomatoid diseases, pulmonary changes secondary to trauma, and collagen and cardiovascular diseases.

Consideration is given to industrial hazards and to assaying lung diseases due to exposure to noxious fumes, gases, and dusts, and to the pathology, symptomatology, diagnosis, and treatment of foreign bodies in the air and food passages.

The volume is intended for the practicing physician, the chest specialist, and the medical student.

A SYNTHESIS OF HUMAN BEHAVIOR: An integration of thought processes and ego growth. By Joseph C. Solomon, Assistant Clinical Professor of Psychiatry, University of California; Consultant, Mount Zion Hospital, San Francisco, California. Cloth. Pp. 265. Price $5.50. Grune & Stratton, 381 Fourth Ave., New York 16, 1954.

This book represents a different approach to a currently popular topic. The material of the book is old, but its arrangement is unique. By using the development of the ego as a guide, the author divides the life span of human beings into ten steps: (1) maturation phase, (2) infancy (nursing situation—teething), (3) toddler (training and indoctrination), (4) oedipal age, (5) latency (prepubertal), (6) pubertal, (7) young adult, (8) mature adult, (9) middle years (climacteric), (10) senescence. Each phase is considered in terms of the psychosexual aspects, principal external objects, mature ego aspects, superego reactions, and neurotic ego traits. As might be concluded it is a study of developmental psychology in terms of psychoanalytic tenets.

The book is well written in language that any physician can follow with interest. There are enough case illustrations to make clear the practical aspects. Its contents are applicable in the study of any behavioral problem, but probably those of childhood and youth can be benefited most. Here is a portrayal of what to expect if certain situations are not corrected. The book can be recommended.

THOMAS J. MEYERS, Ph.D., D.O., F.A.C.N.

The Ciba Collection of Medical Illustrations, Volume I: REPRODUCTIVE SYSTEM. A Compilation of Paintings on the Normal and Pathologic Anatomy, prepared by Frank H. Netter, M.D. Cloth. Pp. 286. Price $13.00. Ciba Pharmaceutical Products, Summit, N. J., 1954.

In the first volume of The Ciba Collection, the stated intention was "to portray in 'desirable detail' the major anatomy and pathology of all the systems comprising the human organism and to devote a separate volume to each system." Although the scope of the second volume is broader than that of the first, the volume on the reproductive system continues to fulfill the intentions previously decided upon.

In preparing the book, Dr. Netter and the editor, Dr. Ernst Oppenheimer, collaborated with a group of well-known teaching physicians. Contained in the volume are 233 full-color plates with accompanying descriptive text. The craftsmanship of Dr. Netter's illustrations cannot be described; the pictures clearly show that they result from the unique combination of physician and artist.

The volume presents the anatomy, physiology, and pathology of the male and female reproductive tracts. The decision to combine the treatises on the female and male reproductive systems into one volume was made in order to integrate knowledge of the two tracts. The description of anatomy of the entire system precedes the description of the anatomy of the individual organs. The work is not intended as a text; it is intended to supplement rather than to replace standard reference works.

THE TECHNIQUE OF PSYCHOTHERAPY. By Lewis R. Wolberg, M.D., Director, Postgraduate Center for Psychotherapy; Associate Clinical Professor of Psychiatry, New York Medical College. Cloth. Pp. 869. Price $14.75. Grune & Stratton, Inc., Medical Publishers, 381 Fourth Ave., New York 16, 1954.

This is a rather mammoth work, appearing even larger than its 869 pages. It is an ambitious effort, and the author has succeeded, to a degree, in producing an encyclopedia of psychotherapy.

The book is divided into five parts; (1) the scope, types and general principles of psychotherapy, (2) the beginning phase of treatment, (3) the middle phase of treatment, (4) the terminal phase of treatment, and (5) special aspects. This division is further clarified by a categorization of the varying views and methods of psychiatric treatment. This feature alone makes the book of great practical value, for it brings order out of what often appears as chaos. For example, therapy is separated into supportive, insight with reeducative goals, insight with reconstructive goals, and brief psychotherapy. Supportive therapy is further divided into guidance, environmental manipulation, externalization of interests, reassurance, prestige suggestion, pressure and coercion, persuasion, emotional catharsis, muscular relaxation, hydrotherapy, shock therapy and drug therapy, brain surgery, inspirational group therapy, and music therapy. The other categories are detailed equally with considerable explanation of just what each means. A very useful chapter is included which describes in simple language the bases of the different schools of psychiatric thought, notably those of Freud, Jung, Adler, Ferenczi, Rank, Stekel, Reich, Rado, Horney, and Sullivan. He then discusses who can do psychotherapy, what equipment is necessary and some general principles. From this point on the technics and procedures are illustrated by verbatum quotation of actual sessions. A case is carried through nine sessions, giving the detailed responses, to illustrate the handling of a problem. There is then appended a recommended reading list and sample forms for the conduct of psychotherapeutic practice, and the recording of progress.

The book is a gold mine of suggestions, for almost every conceivable situation and problem is discussed. For reference and study, it is a valuable aid, but one must remember, of course, that psychotherapy cannot be learned from a book. However, after mastering the elements, a book such as this becomes a right arm. It is to be highly recommended.

THOMAS J. MEYERS, Ph.D., D.O., F.A.C.N.

SURGERY OF THE HEART. By Charles P. Bailey, M.D., M.Sc. (Med.), L.L.D. (Hon.), F.A.C.S., F.C.C.P., F.I.C.S., Professor and Head of the Department of Thoracic Surgery, Hahnemann Medical College and Hospital, Philadelphia, Pa.; Director of Thoracic Surgery, Deborah Sanatorium, Browns Mills, N. J.; Director, Bailey Thoracic Clinic, Philadelphia, Pa.; Thoracic Surgeon: Doctors Hospital, Inc., Philadelphia; Abington Memorial Hospital, Abington, Pa.; Philadelphia General Hospital, Philadelphia; United States Veterans Hospital, Philadelphia, Pa. Cloth. Pp. 1062, with illustrations. Price $25.00. Lea & Febiger, South Washington Square, Philadelphia 6, 1955.

It is only recently that cardiac surgery has developed into a specialty. The great strides that have been made in this field are the result of the work of many pioneers, and one of the most outstanding of these is Dr. Bailey. This text presents many of his own technics as well as those of others.

The author had three purposes in writing this book: (1) to present in adequate concise form the concepts and surgical technics developed at the Bailey Thoracic Clinic or proved sound by him and his associates, (2) to stimulate undergraduate and graduate medical students by pointing out the surgical possibilities of the various conditions discussed, and (3) to acquaint practicing physicians with the concepts, indications, contraindications, operative risks, and the likelihood of attaining satisfactory clinical results in surgery of various cardiac conditions.

The first six chapters consider the development of cardiac surgery, anesthesia, cardiac resuscitation, hypothermia, the heart-lung machine, and exploratory surgery of the heart.

The next ten chapters are devoted to surgery for congenital heart disease—patent ductus arteriosus, aortic septal defects, coarctation of the aorta, vascular compression of the esophagus and/or trachea, pulmonic stenosis, atrial septal defects and anomalous pulmonary venous drainage, ventricular septal defects, transposition of the great arteries, diverticulum of the heart, and pericardial cysts.

The final two thirds of the text consists of descriptions of surgery for acquired heart disease, including trauma to the heart; pericarditis; mitral, aortic, and tricuspid stenosis and regurgitation; multivalvular lesions; hydatid disease of the heart; tumors of the heart; coronary arterial disease; and aneurysms.

In each chapter there is a discussion of normal anatomy and physiology, pathologic anatomy, and pathophysiology, in addition to the presentation of the surgical technics.

This extensively illustrated book will prove useful to surgeons, cardiologists, internists, general practitioners, and pediatricians, and to undergraduate and graduate medical students.

THORACIC SURGERY. By Richard H. Sweet, M.D., Associate Clinical Professor of Surgery, Harvard University Medical School. Ed. 2. Cloth. Pp. 381, with illustrations. Price $10.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1954.

The specialized field of thoracic surgery has been brought up to date in this second edition to include new technics and alterations of technics that have gained acceptance since the original text was published in 1950. Innumerable changes and additions have been made. Some changes in the illustrations have improved the excellent quality found in the first edition: there are entirely new drawings to supplant discarded ones, others have been altered to increase their value, and appropriate illustrations have been added to accompany new sections. There are also new photographs showing instruments that have been added to the armamentarium of the thoracic surgeon.

As in the original text, the revised edition covers every aspect of the specialty. Arranged upon a regional anatomic basis, the book opens with a chapter on the surgical anatomy of the thorax, which is followed by introductory chapters on general technical considerations and thoracic incisions. The technics involved in the major and more common thoracic operations are then discussed and thoroughly explained. Also included is a chapter on abdominal operations performed through thoracic incisions.

The surgeon, surgical resident, and those interns and medical students who intend to specialize in thoracic surgery will find this book a worth-while addition to their libraries.

The Clinical Significance of DISTURBANCES IN THE DELIVERY OF SWEAT. By Marion R. Sulzberger, M.D., F.A.C.P., and Franz Herrmann, M.D., Department of Dermatology and Syphilology, New York University Post-Graduate Medical School; Skin and Cancer Unit of University Hospital, New York University-Bellevue Medical Center. Paper. Pp. 212, with illustrations. Price $6.75. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

This book, one of the "American Lectures in Dermatology," is certainly one of the most excellent of this series. While books are current only to their date of publication, this one will have much to offer for a long time in background reading. The first section deals with investigative approaches, the second with pathogenesis and characteristics of disturbances in the delivery of sweat, and the third with the most common disturbances.

Hyperhidrosis in its many aspects is discussed, and we could not but underline one section that constitutes basic information for pediatricians and for the mothers with whom they deal. Under the heading, "Pseudo-hyperhidrosis," it is stated that "the influence of elevated environmental temperature is physiologically increased during sleep." This is so applicable to the sweating of infants and the efforts of indulgent mothers to keep them warm.

Of great interest to a dermatologist is the discussion of the effect of Atabrine on sweating. Now that this drug is being used in lupus erythematosus great caution must be used to prevent the occurrence of atrophy of the sweat glands.

The effects of aluminum salts on the eccrine and apocrine glands are considered in detail. The authors' opinion is somewhere between the claims of cosmetic manufacturers and the antialuminum-salts group. Plates of pathologic sections and diagrammatic drawings are used to good advantage in explanations.

The problem of miliaria and its type depending on depth of obstruction in the sweat gland is enlightening and of interest to the obstetrician and pediatrician.

The discussion of chromidrosis (colored sweat), hemathidrosis, and urhidrosis is of less importance clinically and socially than that of bromidrosis (abnormal odor). The causes and treatment of this disease are detailed.

Therapy and the effect of drugs in general on sweat mechanisms are comprehensively reviewed. A most extensive and detailed bibliography completes the book. Needless to say the reviewers think the book worth every physician's time to read.

A. P. Ulbrich, D.O.
D. Koprince, D.O.

ABDOMINAL OPERATIONS. By Rodney Maingot, F.R.C.S., Eng., Surgeon to the Royal Free Hospital, London, and to the Southend General Hospital. Ed. 3. Cloth. Pp. 1580, with illustrations. Price $24.50. Appleton-Century-Crofts, 35 W. 32nd St., New York 1, 1955.

Advances in surgery since 1948 have necessitated a new edition of this text. Sixty-three chapters have been revised, and eleven have been added. Sixteen new contributors have considered certain specialized subjects, and more than 400 new illustrations have been included.

Presented in detail are considerations of the technics of modern abdominal operations. The choice of operation in the individual case is discussed, as are the difficulties and dangers arising during the operation, the preoperative and postoperative treatment, complications and their management, the immediate and long-term results of the operation, and the clinical, pathologic, and other aspects of destructive lesions of the abdominal viscera.

The operative fields covered include the stomach and duodenum, spleen, pancreas, gallbladder and bile ducts, liver, vermiform appendix, peritoneum, mesentery and omentum, intestines, diaphragmatic hernia, portal hypertension, acute phlegmons of the gastrointestinal tract, abdominal actinomycosis, pelvic exenteration, postoperative chest complications, and fluid, electrolyte, and nutritional problems.

The work is intended for the abdominal surgeon, for the general surgeon specializing in abdominal surgery, for resident surgeons, and for the senior student contemplating specialization in surgery.

PRINCIPLES OF INTERNAL MEDICINE. Edited by T. R. Harrison, Raymond D. Adams, William H. Resnik, Paul B. Beeson, George W. Thorn, and M. M. Wintrobe. Ed. 2. Cloth. Pp. 1703, with illustrations. Student price, 1 Vol. Ed., $16.00. Professional price, 2 Vol. Ed., $21.00. The Blakiston Company, 575 Madison Ave., New York 22, 1954.

To attempt to achieve a balance between the art and science of medicine, to present material which deals with practical problems of internal medicine today, and to integrate that material with the basic principles which will probably determine the medical practice of tomorrow were the aims of the editors of this book.

The text consists of nine parts—the first of which deals with introductory material entitled "The Physician and the Patient." In order follow considerations of the cardinal manifestations of disease; biologic considerations; reactions to stress and to antigenic substances; general care of the patient; metabolic and endocrine disorders; disorders due to chemical and physical agents; diseases due to biologic agents; diseases associated with reactions to stress and to antigenic substances; and diseases of organ systems. The sequence which is followed is essentially that used in teaching medical students.

Much of the material in this second edition has been rewritten or revised to bring it up to date. A series of chapters has been added which deals with the more common manifestations of disordered nervous system function because so many constitutional diseases have symptoms referred to the nervous system.

The work is intended primarily for students and practitioners who want a presentation of the important scientific principles necessary for rational understanding of the development, evolution, and management of internal diseases.

THE VOICE OF NEUROSIS. By Paul J. Moses, M.D., Assistant Clinical Professor in Charge of Speech and Voice Section, Division of Otolaryngology, Stanford University School of Medicine, San Francisco, California. Cloth. Pp. 131. Price $4.00. Grune & Stratton, 381 Fourth Ave., New York 16, 1954.

The keynote of this work is expressed in the first sentence: "whoever diagnoses neurosis is consciously or unconsciously affected by the patient's voice. The man who is afraid will show it in his voice as well as in his posture, gait, gestures and through what he says. Voice is the primary expression of the individual, and even through voice alone the neurotic pattern can be discovered."

The author has been developing and perfecting the analysis of the personality from the voice for the past 15 years. Such an analysis means, first, resolving an entity (the voice) into its constituent elements so that they reveal their relations to one another and to the whole; second, that these elements must be described in measurements or other unambiguous verifiable terms; third, that the influences which shape any observed variations must be traced; and, finally, that these parts be integrated into meaningful patterns. For example, the neurotic voice reveals typical symptoms of fixation and regression through vocal patterns belonging to earlier phases in its development, whereas the schizophrenic voice has a marked archaic character with primordial attributes.

Dr. Moses analyzes his method of acoustic measurement—which he claims can be done with marked objective accuracy. Speech must have universal rhythm validity as well as semantic validity to be a useful means of communication. Voice without melody is truly meaningless; in other words, it must express feeling as well as reasoning.

The last two chapters as given over to an interpretation of the neurotic personality from the voice.

This is a remarkable book; it is one of those rare ones that incorporates a "new" idea and a "new" approach to an old problem. The author has given a practical understandable technic for the evaluation of an everyday universal, interpersonal series of problems. It is a book that can be most highly recommended.

Thomas J. Meyers, Ph.D., D.O., F.A.C.N.

FUNDAMENTALS OF ANESTHESIA. Prepared under the Editorial Direction of the Consultant Committee for Revision of Fundamentals of Anesthesia, a publication of the Council on Pharmacy and Chemistry of the American Medical Association. Ed. 3. Cloth. Pp. 279. Price $6.00. W. B. Saunders Company, West Washington Square, Philadelphia, 1954.

This third edition is practically a new book, for its content has not only been widely rearranged but also largely rewritten. This book is designed as an outline or guide in the teaching of the fundamentals of anesthesia. The basic essentials are presented briefly and perforce dogmatically, but with sufficient explanatory text. The neophyte in the field of anesthesiology who masters the basic principles presented here will become adept in his technic.

This edition is better illustrated than the older ones, and it retains the display in strategic positions of well-known aphorisms pertaining to anesthesiology.

The many omissions in the text, which are readily apparent to the specialist, in no way impair the value of the book for students in the field of anesthesiology. Every one of them would do well to have this book in his library.

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it may well be that mealtime has become something "to get over with" rather than a pleasurable pastime. Introduction of a variety of foods and new menu interest can often play a big part in bringing a balky patient back to the table. Gerber offers 4 Cereals, over 60 Strained and Junior (minced) Foods to give you greater latitude in specifying a well-balanced, nutritionally-sound diet. PLUS:

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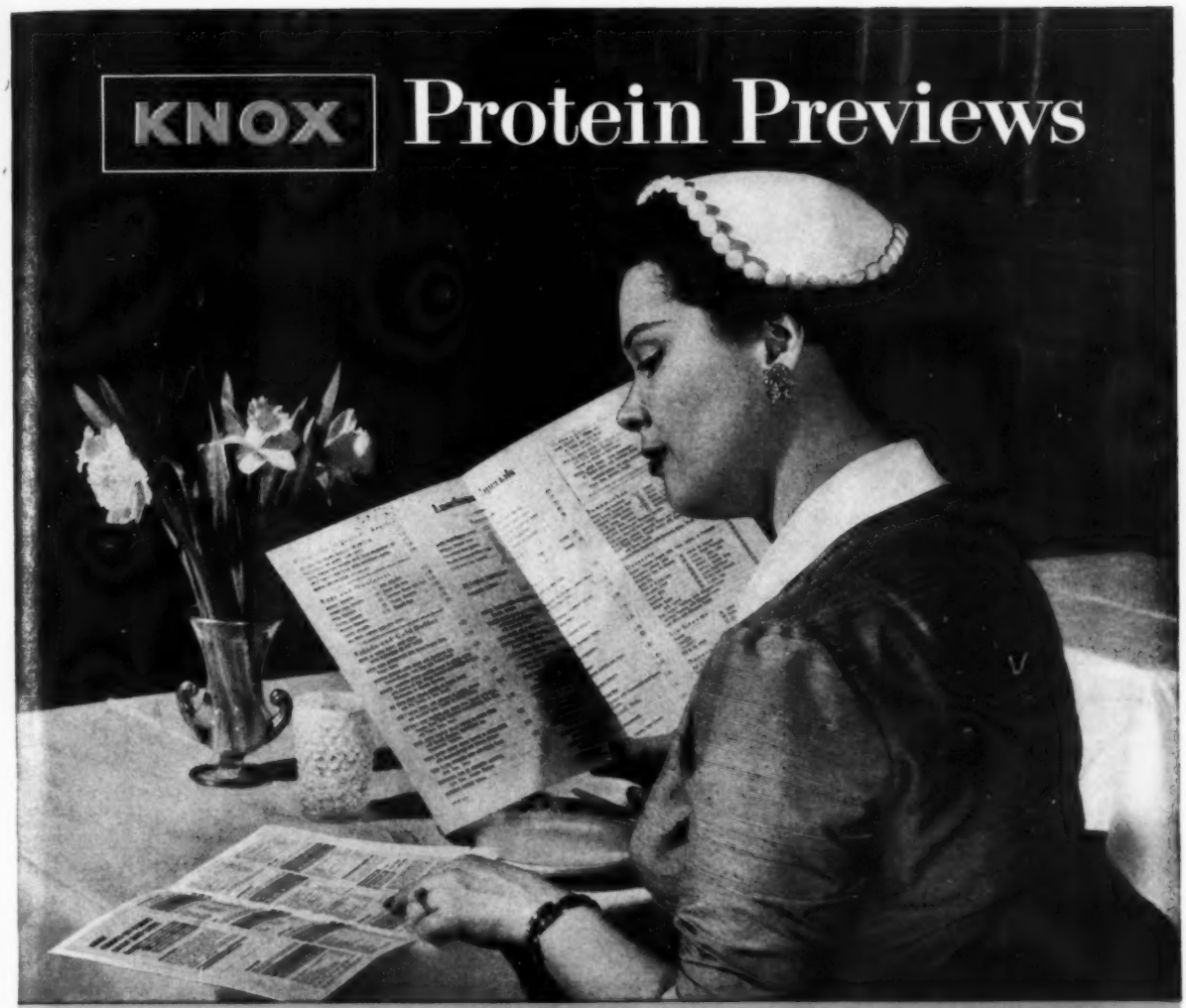

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1. Developed by the U. S. Public Health Service assisted by committees of The American Diabetes Association, Inc. and The American Dietetic Association.

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Please send me_____copies of the new, color-coded "choice-of-foods diet list"-chart.

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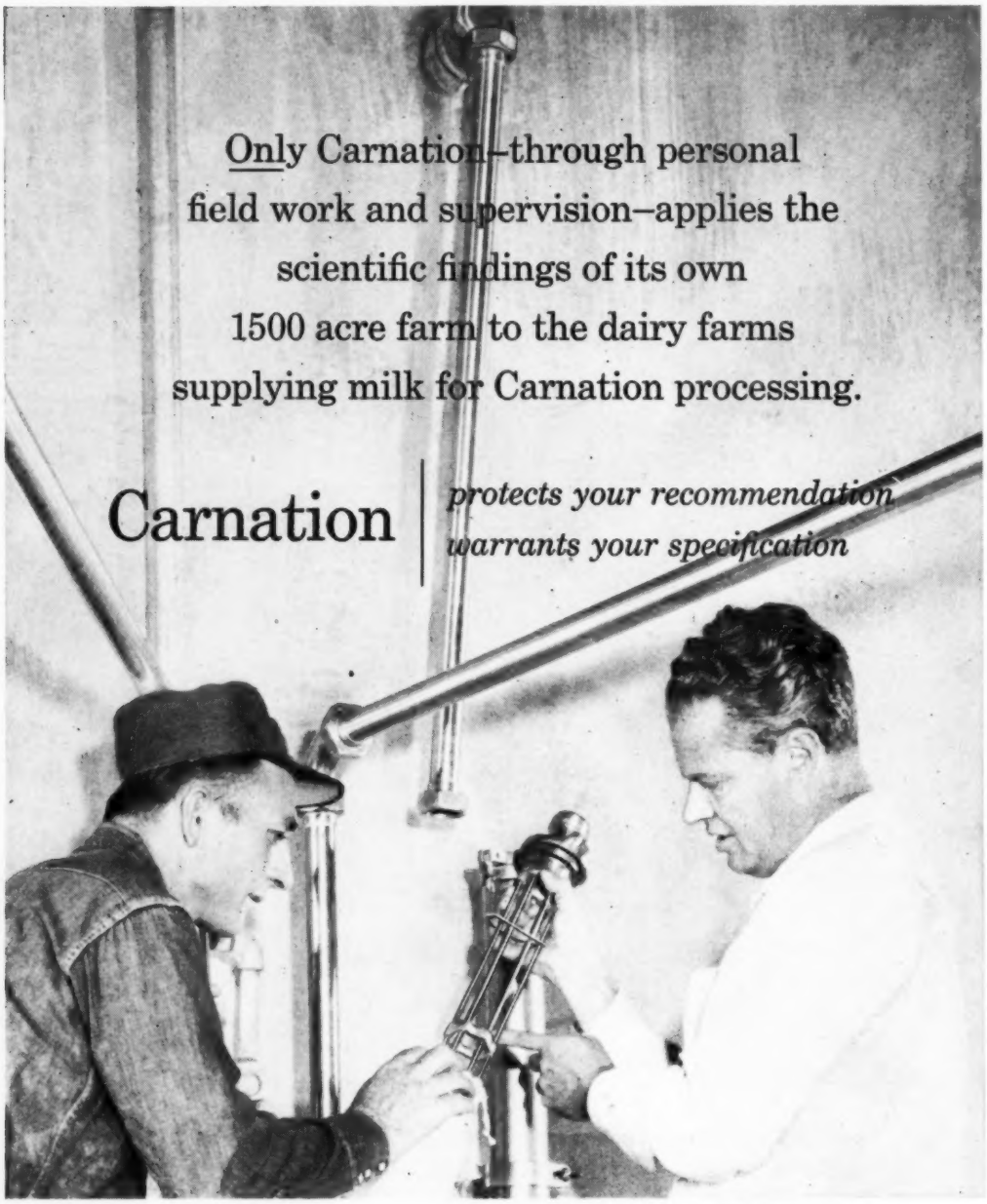
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field work and supervision–applies the
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protects your recommendation
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Brand of oxytetracycline with vitamins
CAPSULES 250 mg.

®Tetracyn-SF
Brand of tetracycline with vitamins
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ORAL SUSPENSION (fruit flavored)
125 mg./5 cc. teaspoonful

The minimum daily dose of each antibiotic *(1 Gm. of Terramycin or Tetracyn, or 600,000 units of penicillin)*
Stress Fortifies the patient *with the stress vitamin formula* **recommended by the National Research Council:**

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Thiamine mononitrate	10 mg.	Vitamin B_{12} activity	4 mcg.
Riboflavin	10 mg.	Folic acid	1.5 mg.
Niacinamide	100 mg.	Menadione (vitamin K analog)	2 mg.
Pyridoxine hydrochloride	2 mg.		

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THE many advantages of enriched bread in special diets are all too seldom recognized or appreciated. For this reason, the physical, physiologic, nutritional, and dietetic values of enriched bread warrant detailed enumeration.

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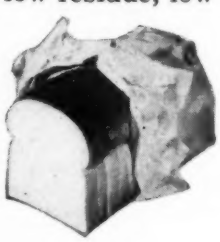
For these many reasons, enriched bread enjoys a prominent place in a great variety of special diets—soft, low residue, low purine or cholesterol diets, general hospital diets, as well as diets low or high in carbohydrates, in proteins, or in calories.



The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

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"It is questionable if any of the common bases used in ointments (e.g., fixed oils, fats, lanolin or petrolatum) facilitate the absorption through the normal skin of drugs incorporated in them," writes Grollman[2].

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Apply daily after a mild soap bath and thorough drying. A thin, invisible, economical film suffices. No bandages required. After one week, adjust to patient's progress.

RIASOL is supplied in 4 and 8 fld. oz. bottles at pharmacies or direct.

1. Vickers, H. R., in *Psoriasis*, in *Modern Practice in Dermatology*, 1950, pp. 241-250.
2. Grollman, A., *Pharmacology and Therapeutics*, 1954, p. 558.

Before using RIASOL

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Especially whenever tender skin is irritated by heat, moisture or chafing, AMMENS Powder gives a prompt feeling of soothing comfort.

AMMENS' starch granules, evenly dispersed in talc, provide an absorbent coating on irritated skin. Oxyquinolin and zinc oxide, blended in the powder, help protect macerated crevices against bacterial invasion.

For skin comfort—especially following strenuous work or play, or in hot weather—keep a can of AMMENS close at hand.

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Multivitamins for children 2 to 10

UNIQUE SOFTAB† FORM

melts in the mouth

PLEASANT TASTING

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One tablet contains:

A	5000 USP Units
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C	60 mg.
B_1	2 mg.
B_2	2 mg.
B_6	1 mg.
B_{12}	5 mcg.
Calcium Pantothenate	3 mg.
Niacinamide	10 mg.

DOSE: 1 TABLET DAILY

SUPPLIED:

BOTTLES OF 50 and 100 TABLETS

"CHAIN REACTION" THERAPY...

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Vasodilator—

—rapidly oxygenates tissues and relieves ischemia, a major source of pain.

INDICATIONS:
Peripheral vascular disorders
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Why risk side effects from one antihistamine when a combination of three antihistamines means greater safety.

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Available in Capsules and exceptionally palatable fruit-flavored Syrup, (half-strength) for children.
Each capsule contains:

Pyrilamine maleate	10 mg.
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Syrup: Each 5 cc contains half of the above.

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FOR MANAGEMENT OF
PERIPHERAL VASCULAR DISORDERS

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TABLETS

— delivers coenzymes to metabolize accumulations of toxic substrates resulting from inadequate oxidation.

— thus provides the safe, metabolic approach to control of pain caused by inadequate peripheral circulation and impaired tissue metabolism.

EACH VASTRAN TABLET CONTAINS:

Nicotinic acid	50 mg.
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Riboflavin	5 mg.
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dosage:
One tablet three times daily, preferably on an empty stomach.

supplied:
Bottles of 100 and 500 scored tablets.

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New 1955 Book

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Practical Management of Disorders of the

LIVER, PANCREAS and BILIARY TRACT

By John Russell Twiss, M.D., F.A.C.P.
Assistant Professor of Clinical Medicine, New York University Post-Graduate Medical School
and Elliot Oppenheim, M.D., F.A.C.P.
Assistant Professor of Clinical Medicine, New York University Post-Graduate Medical School
And Contributors

New. Doctors who want a sound and authoritative guide in the diagnosis and management of commonly met disorders of the liver, pancreas and biliary tract will find it in this comprehensive book. Although the gallbladder and extrahepatic ducts, liver and pancreas are considered separately, the importance of clinical integration is stressed throughout. A fundamental purpose of the book is to facilitate and correlate diagnostic procedures and appropriate methods of management.

The greatest emphasis throughout is on medical diagnosis and treatment. The surgical aspects are considered in relation to principles and indications for surgery, rather than actual operative procedures. Instructions on cholecystography, cholangiography, needle biopsy, and other diagnostic and laboratory tests; as well as therapeutic diets, are included. Osteopathic physicians, internists, gastroenterologists, and surgeons, as well as clinical and investigative workers, will find this book to be a most practical and useful presentation.

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antibiotics *Stress Fortified* with vitamins include:

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Brand of oxytetracycline with vitamins
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125 mg./5 cc. teaspoonful

The minimum daily dose of each antibiotic *(1 Gm. of Terramycin or Tetracyn, or 600,000 units of penicillin)*
Stress Fortifies the patient *with the stress vitamin formula* **recommended by the National Research Council:**

Ascorbic acid, U.S.P.	300 mg.	Calcium pantothenate	20 mg.
Thiamine mononitrate	10 mg.	Vitamin B_{12} activity	4 mcg.
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Niacinamide	100 mg.	Menadione	
Pyridoxine hydrochloride	2 mg.	(vitamin K analog)	2 mg.

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PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc.*, Brooklyn 6, N. Y.

Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Ninth Annual Convention, College of Osteopathic Physicians and Surgeons and Biltmore and Statler Hotels, Los Angeles; Convention Instruction Courses, July 5-16; Convention Teaching Sessions, July 18-22. Program Chairman, W. Donald Baker, 3450 W. 43rd St., Los Angeles 8.

Academy of Applied Osteopathy, annual meeting, Statler Hotel, Los Angeles, July 22-23. Program Cochairmen, Margaret H. Raffa, 5009 Central Ave., Tampa 3, Fla., and Paul K. Theobald, 1419 Broadway, Oakland 12, Calif. Secretary, Margaret W. Barnes, Box 1345, Carmel, Calif.

American College of Osteopathic Internists, annual meeting, Sheraton-Cadillac Hotel, Detroit, October 20-22. Program Chairman, Ward E. Perrin, Chicago Osteopathic Hospital, 5250 S. Ellis Ave., Chicago 15. Secretary, Glennard E. Lahrson, 460 Staten Ave., Oakland 10, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Neil House Hotel, Columbus, Ohio, February 6-9. Program Chairman, Richard E. Eby, 1247 N. Park Ave., Pomona, Calif. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

American College of Osteopathic Surgeons, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, Arthur M. Flack, 135 S. 17th St., Philadelphia 3. Executive Secretary, Orel F. Martin, Box 474, Coral Gables 34, Fla.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Statler, Washington, D. C., October 29-November 2. Program Chairman, Karl P. B. Madsen, 460 Staten Ave., Oakland 10, Calif. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8.

American Osteopathic Academy of Physical Medicine and Rehabilitation, annual meeting, Los Angeles, July 23. Program Chairman, W. Irvin Harner, 302 W. Hadley St., Whittier, Calif. Secretary, John A. Schuck, 1739 Griffin Ave., Los Angeles 31.

American Osteopathic College of Anesthesiologists, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, Harry J. Petri, Jr., 5 Deering St., Portland 3, Maine. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo.

American Osteopathic College of Radiology, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, A. G. Reed, 212 Pythian Bldg., Tulsa 3, Okla. Secretary, F. A. Turfler, Jr., South Bend Osteopathic Hospital, 118 S. William St., South Bend 2, Indiana.

American Osteopathic Hospital Association, annual meeting, Hotel Statler, Washington, D. C., October 30-November 2. Program Chairman, Mrs. Alixe P. Nuzum, Des Moines General Hospital, Des Moines, Iowa. Executive Secretary, Mr. R. P. Chapman, 1013 Kahl Bldg., 326 W. Third St., Davenport, Iowa.

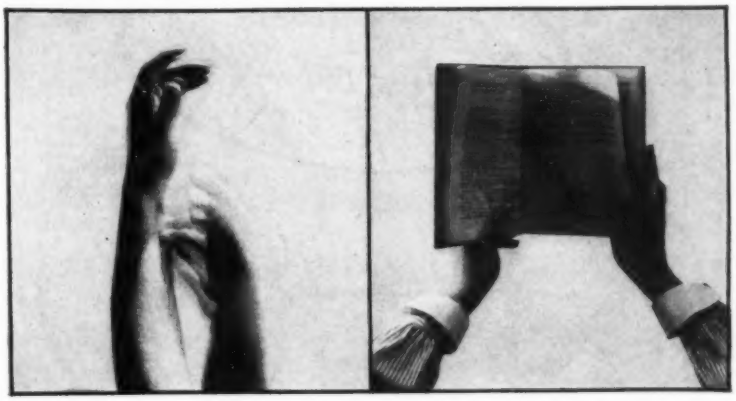
Canadian Osteopathic Association, annual meeting, Mount Royal Hotel, Montreal, October 27-29. Program Chairman, A. E. Wilkinson, 616 Medical Arts Bldg., Montreal 25. Secretary, Miss Joyce C. Currie, 609 Medical Arts Bldg., Montreal 25.

Hawaii, annual meeting, Reef Hotel, Honolulu, July 25-27. Program Chairman, C. W. Wyman, 417 National Bldg., Honolulu 13. Secretary, Joseph W. Stella, 38 Young Hotel Bldg., Honolulu 13.

Kentucky, annual meeting, Brown Hotel, Louisville, October 12-13. Program Chairman, Henry P. Lawrence, Hawesville. Secretary, Harold D. Benteen, 2048 Winchester Ave., Ashland.

Maine, annual meeting, Samoset Hotel, Rockland, June 16-18. Program Chairman, Michael A. Longo, 142 Pine St., Bangor. Midyear meeting, Eastland Hotel, Portland, December 2-3. Secretary, Roswell P. Bates, 72 Main St., Orono.

Of two patients with poison ivy...

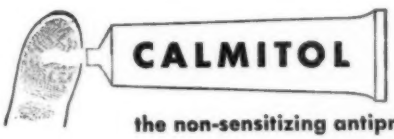


one aggravates the dermatitis venenata by vicious scratching; the result: excoriation and infectious eczematoid dermatitis.

the other is not disturbed by itching. The dermatitis venenata is permitted to clear rapidly and without annoying complications.

Calmitol makes the difference:

Nonsensitizing and free of the dangers of "rebound dermatosis," Calmitol is "preferred"[1] by physicians for its safe and prolonged antipruritic action.



the non-sensitizing antipruritic

1½ oz. tubes and 1 lb. jars

1. Lubowe, I. I.: New York State J. Med. 50:1743, 1950.

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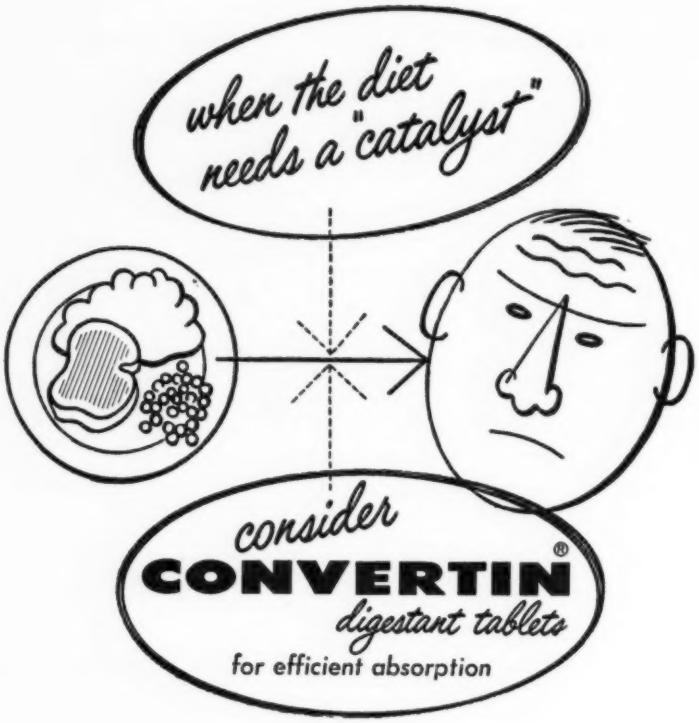

When your geriatric, dyspeptic, underweight, or gallbladder patient doesn't respond to diet, the cause is frequently an inability to *utilize food.*

CONVERTIN furnishes the dietary catalysts necessary for efficient absorption in these individuals.

The specially layered construction of CONVERTIN provides selective release of ingredients to assure efficient absorption in the stomach and small intestine.

Each CONVERTIN Tablet provides:
a sugar-coated outer layer of:
Betaine Hydrochloride.....130.0 mg.
(Provides 5 minims Diluted Hydrochloric Acid U.S.P.)
Oleoresin Ginger.....1/600 gr.
Surrounding an enteric-coated core of:
Pancreatin.....62.5 mg.
(Equiv. 250 mg. U.S.P.)
Desoxycholic Acid.....50.0 mg.
DOSAGE: One or two tablets with or just after meals.
SUPPLIED: In bottles of 84 and 500 tablets.

B. F. ASCHER & COMPANY, INC.
Ethical Medicinals
KANSAS CITY, MISSOURI

Massachusetts, annual meeting, Hotel Somerset, Boston, January 21-22. Program Chairman, Everett L. Pierce, Pleasant St., East Dennis. Executive Secretary, Mrs. Gladys M. Stockdale, 524 California St., Newtonville.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, October 3-5. Program Chairman, Harold W. Laidlaw, McCoy-Laidlaw Clinic, Bad Axe. Executive Secretary, Mr. Harve Lamont Smith, 81 Glendale, Highland Park 3.

Missouri, annual meeting, Continental Hotel and Municipal Auditorium, Kansas City, September 27-29. Program Chairman, J. Edward Sommers, 6504A Delmar Blvd., St. Louis 5. Executive Secretary, Mr. Paul D. Adams, 325 E. McCarty St., Jefferson City.

New Jersey, midyear meeting, Stacy-Trent Hotel, Trenton, September 18. Annual meeting, Essex House, Newark, March 10-11. Program Cochairmen for both meetings, George W. Northup, 104 S. Livingston Ave., Livingston, and Harry A. Sweeney, 25 S. Illinois Ave., Atlantic City. Executive Secretary, Mr. I. J. Tecker, 21 Hancock St., Riverside.

New York, annual meeting, Hotel Statler, New York City, October 14-15. Program Chairman, Walter Streicker, 7 Plaza St., Brooklyn 17. Secretary, Robert E. Cole, 417 S. Main St., Geneva.

North Carolina, annual meeting, Battery Park Hotel, Asheville, October 20-22. Secretary and Program Chairman, S. Dales Foster, 710 Public Service Bldg., Asheville.

Northwest Osteopathic Convention, annual meeting, Hotel Multnomah, Portland, Ore., June 13-16. Program Chairman, Russell F. Kenaga, Sr., 2265 W. Burnside St., Portland 5.

Oklahoma, annual meeting, Hotel Tulsa, Tulsa, November 8-10. Program Chairman, Carl R. Samuels, S. Adair St., Box 44, Pryor. Executive Secretary, Mr. Walter L. Gray, 210-12 Braniff Bldg., Oklahoma City.

Oregon: See Northwest Osteopathic Convention.

Osteopathic College of Ophthalmology and Otorhinolaryngology, annual meeting, Continental Hotel, Kansas City, Mo., July 24-27. Program Chairman, H. Mahlon Gehman, 1818 Pine St., Philadelphia 3. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15, Ohio.

Pennsylvania, annual meeting, Bellevue-Stratford Hotel, Philadelphia, September 30-October 2. Program Chairman, George S. Esayian, 113 N. Ambler St., Quakertown. Executive Secretary, Mr. George W. Thomas, 1941 Market St., Harrisburg.

Vermont, annual meeting, Long Trail Lodge, Mendon Mt., Rutland, September 24-25. Program Chairman, Hermon K. Sherburne, Jr., Mead Bldg., Rutland. Clerk, Howard I. Slocum, Battell Block, Middlebury.

Washington, midyear meeting, Washington Athletic Club, Seattle, December 3-4. Program Chairman, Erskine H. Burton, 710 Jones Bldg., Tacoma 2. Secretary, Eugene E. LaCroix, 1747 S. Sheridan Ave., Tacoma 5.
See also Northwest Osteopathic Convention.

State and National Boards

ALABAMA

Examinations June 21-23 at the State Capitol, Montgomery. Applications must be filed by June 20. Address D. G. Gill, M.D., secretary, Board of Medical Examiners, State Office Bldg., Montgomery 4.

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Basic science examinations September 20 at the University of Arizona, Tucson. Applications must be filed 2 weeks prior to the examination. Address Herbert D.

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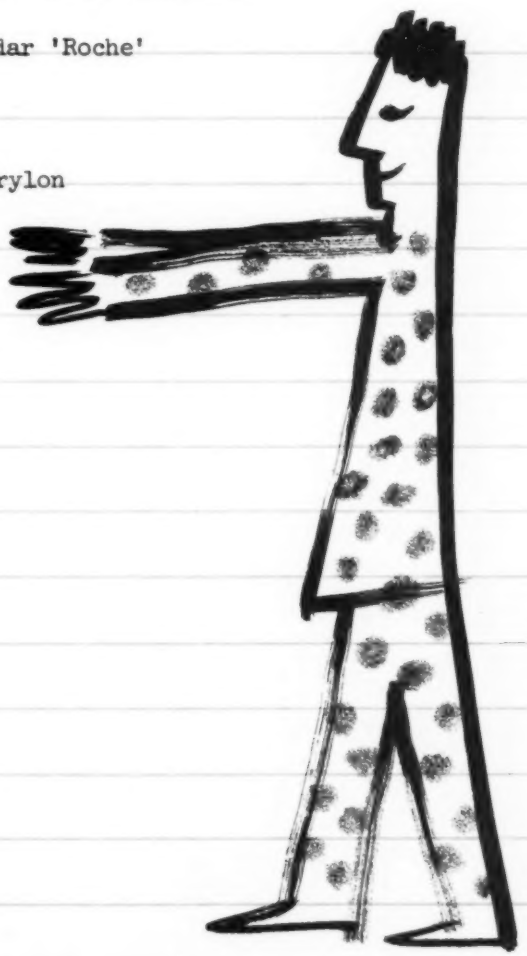

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Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Basic science examinations in September. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

DELAWARE

Examinations July 12-14. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

GEORGIA

Examinations July 5 at Atlanta. Address Robert K. Glass, D.O., secretary, Board of Osteopathic Examiners, 834-5 Forsyth Bldg., Atlanta 3.

ILLINOIS

Examinations in June in Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations July 12 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

Examinations in June at Topeka. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania Ave., Holton.

MAINE

Examinations June 14-15 at Augusta. Address George Frederick Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Square, Dover-Foxcroft.

MARYLAND

Examinations in June. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 419 No. Charles St., Baltimore 1.

MASSACHUSETTS

Examinations July 12. Address Robert C. Cochrane, M.D., secretary, Board of Registration in Medicine, Room 37, State House, Boston 33.

MISSISSIPPI

Examinations June 27-28 at the Robert E. Lee Hotel, Jackson. Applications must be filed by June 17. Address R. N. Whitfield, M.D., assistant secretary, Board of Health, Jackson.

MONTANA

Examinations in September. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEVADA

Professional examinations June 25 at 210 W. Second St., Reno. Applications must be filed 2 weeks prior to examination. Address Walter J. Walker, D.O., secretary, Board of Osteopathic Examiners, 210 W. Second St., Reno.

Basic science examinations July 5. Address Donald G. Clooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW HAMPSHIRE

Examinations September 14-15 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW JERSEY

Examinations June 21. Address Patrick H. Corrigan, M.D., acting secretary, Board of Medical Examiners, 28 W. State St., Trenton.

NEW MEXICO

Basic science examinations July 17. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P. O. Box 1522, Santa Fe.

NEW YORK

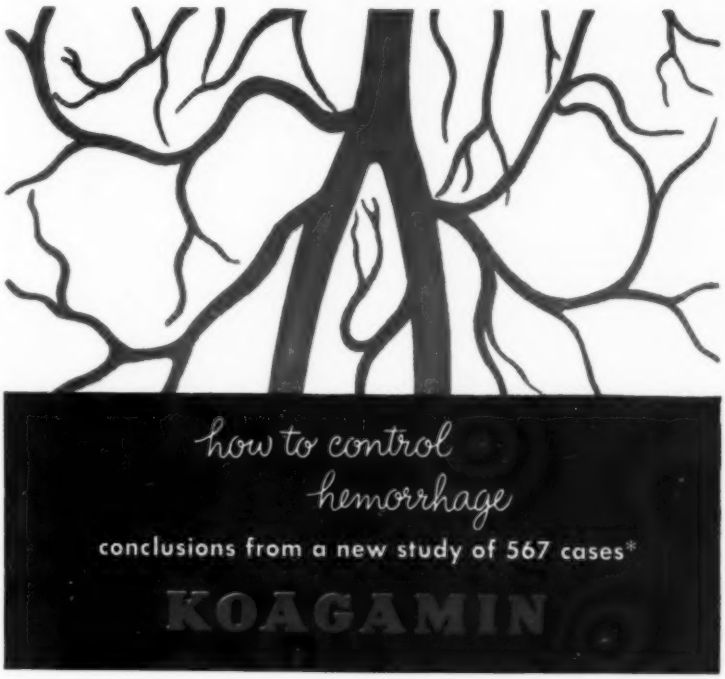
Examinations June 28-July 1. Address John W. Paige, M.D., chief, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

NORTH CAROLINA

Examinations July 1-2 at Raleigh. Reciprocity fee $25.00. No annual registration requirement. Address Frank R. Heine, D.O., secretary, Board of Osteopathic Examination and Registration, 926 Southeastern Bldg., Greensboro.

NORTH DAKOTA

Examinations July 9. Applications must be filed by June 15. Address G. L.



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OHIO

Examinations June 13-15 at Men's Gymnasium, Ohio State University, Columbus. Address H. M. Platter, M.D., secretary, Medical Board, 21 W. Broad St., Columbus 15.

OREGON

Basic science examinations September 10. Address Mr. John R. Richards, secretary, State Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations July 7-8. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations August 31 in Room 366, State Office Bldg., Providence. Applications must be filed approximately 20 days prior to examination. Address Mr. Casey.

SOUTH CAROLINA

Examinations June 21 at Columbia. Address Ernest A. Johnson, D.O., secretary, Board of Osteopathic Examiners, Box 525, Summerville.

SOUTH DAKOTA

Professional examinations July 19-20 at Sioux Falls. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

TENNESSEE

Examinations August 3 at Nashville. Applications must be filed by July 1. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

TEXAS

Examinations June 20-22 in Fort Worth. Address M. H. Crabb, M.D., secretary, Board of Medical Examiners, 1714 Medical Arts Bldg., Fort Worth 2.

VERMONT

Examinations in June. Address Charles D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

WASHINGTON

Professional examinations July 10-13 at Seattle. Applications must be filed 15 days prior to examination. Address Mr. Edward C. Dohm, secretary, Professional Division, Department of Licenses, Olympia.

Basic science examinations July 6-7 at Seattle. Applications must be filed 15 days prior to examination. Address Mr. Dohm.

WEST VIRGINIA

Examinations June 27-28 at the Stonewall Jackson Hotel, Clarksburg. Applications must be filed 10 days prior to examination. Address W. S. Irvin, D.O., secretary, Board of Osteopathy, 202 Broadway St., Berkeley Springs.

WISCONSIN

Professional examinations July 12-13 at Milwaukee. Address Thomas W. Tormey, Jr., M.D., secretary, Board of Medical Examiners, State Office Bldg., 1 West Wilson St., Madison.

Basic science examinations September 23 at the Assembly Chamber, State Capitol, Madison. Applications must be filed by September 15. Address Mr. William H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom St., Ripon.

REREGISTRATION OF OSTEOPATHIC LICENSES

During June—Hawaii, $5.00 for residents, $2.00 for nonresidents. Address Frank O. Gladding, D.O., secretary, Board of Osteopathic Examiners, 504 Kauikeolani Bldg., Honolulu 13.

Before June 30—Delaware, $20.00. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

June 30—Virginia, $1.00. Address K. D. Graves, M.D., secretary, Board of Medical Examiners, 631 First St., S. W., Roanoke.

July 1—Idaho, $10.00. Address D. W. Hughes, D.O., secretary, Board of Os-

teopathic Examiners, 203 Sun Bldg., Boise.

July 1—Kansas, $5.00. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania Ave., Holton.

July 1—Michigan, $5.00. Address P. Ralph Morehouse, D.O., assistant secretary, Board of Osteopathic Registration and Examination, 214 So. Superior St., Albion.

July 1—New Mexico, $5.00. Address H. E. Donovan, D.O., secretary, Board of Osteopathic Examination and Registration, Donovan Osteopathic Hospital, Raton.

July 1—North Dakota, $3.00. Address G. L. Hamilton, D.O., secretary, Board of Osteopathic Examiners, Ringo Bldg., 119 S. Main St., Minot.

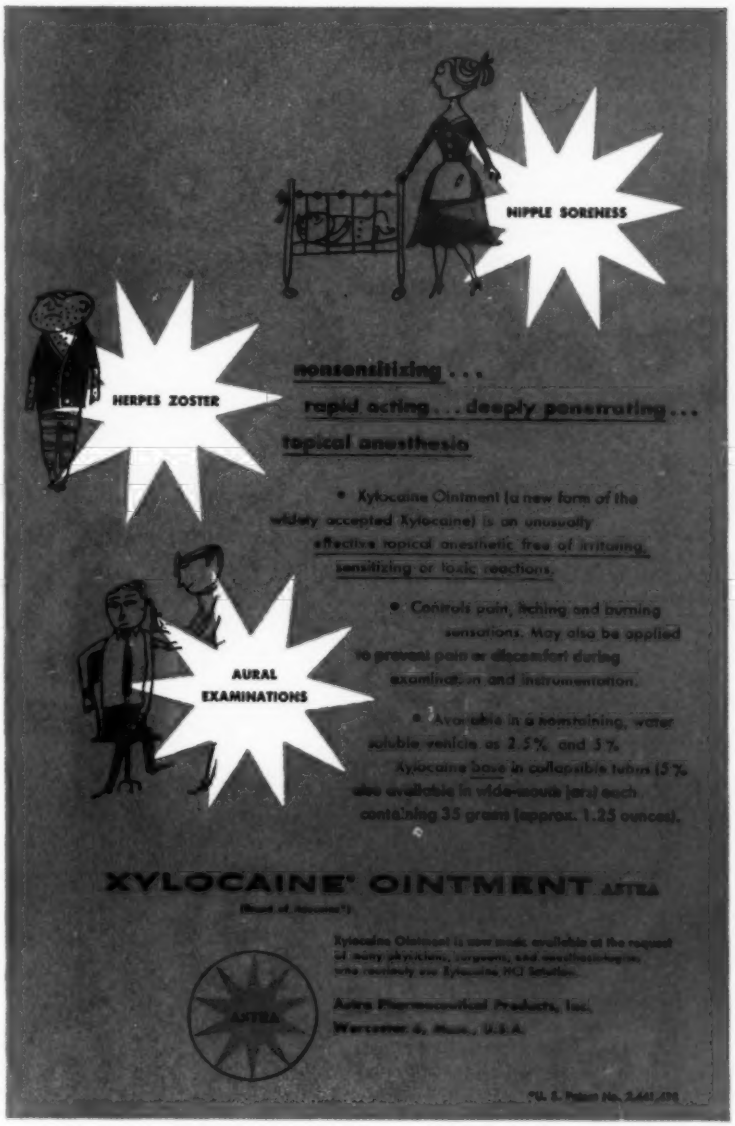
July 1—Oklahoma, $2.00. Address G. R. Thomas, D.O., secretary, Board of Osteopathy, 1100 N. W. 36th St., Oklahoma City 18.

July 1—West Virginia, $2.00. Address W. S. Irvin, D.O., secretary, Board of Osteopathy, 202 Broadway St., Berkeley Springs.

Within 60 days after July 1—Indiana, $5.00 for residents, $10.00 for nonresidents. Address Paul R. Tindall, M.D., secretary, Board of Medical Registration and Examination, 538 K. of P. Bldg., Indianapolis 4.

September 1—Ohio, $2.00. Address H. M. Platter, M.D., secretary, State Medical Board, 21 W. Broad St., Columbus 15.

During September—Nebraska, $2.00. Address C. Eugene Brown, D.O., secretary, Osteopathic Board of Examiners, 1702 Second Ave., Nebraska City.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EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene, medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles; therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

ARTERIOSCLEROSIS STUDIES

Research Progress Report*

Research in cardiovascular health is a prime function of the National Heart Institute, a division of the Public Health Service. Since its founding in 1948, in addition to conducting research in Government laboratories, the institute has granted more than $3.5 million to universities and other institutions to support research related to studies of arteriosclerosis. Most of this amount applies to studies which are still in progress.

The pattern of this research is determined largely by the applicants for grants. The scientists themselves pose the questions which must be answered in advance of further progress in the field.

DEFINITION AND CLASSIFICATION

Relatively few grants have been sought for defining the entity of arteriosclerosis, that condition marked by loss of elasticity, thickening or hardening of the arteries, or atherosclerosis, a specific type of arteriosclerosis characterized by lipoidal and plaquelike changes in the inmost layer of the artery, the intima. One definitive project classifies an entity known as coronary heart disease, from studies of heart muscle lesions. Correlated clinical findings and electrocardiograms are offered to support this diagnosis.

EXPERIMENTAL STUDIES IN PATHOGENESIS

The need to develop experimental techniques is both primary and chronic in the search for contributing factors in arteriosclerosis. The successful use of animals to induce sclerotic processes comparable to those in man began with Anitschkow's work with rabbits in 1908. Since then, experimental studies have employed guinea pigs, rats, dogs, cockerels, chickens, rabbits, and Cebus monkeys. Work supported by the Public Health Service has produced many basic contributions to experimental techniques, involving control of diet, administration of chemicals, action of hormones, transfusion of serums, physical injury, genetic influences, and the effects accompanying aging.

An early need in experimental techniques was to develop simple and reliable methods of inducing the process of arteriosclerosis. Cholesterol, a lipid found in animal fats and oils, whole milk, egg yolk, liver, and certain forms of seafood, was associated with the sclerotic process by Anitschkow. Since diets high in cholesterol do not invariably produce the condition, other contributing forces have been looked for. Low thyroid activity is one. Arteriosclerosis develops in dogs fed a high cholesterol diet supplemented by thiouracil, a chemical which reduces the production of thyroid hormones. The thiouracil does not so affect dogs on a normal diet.

Readings of cholesterol in the serum of dogs are used in efforts to predict the severity of arteriosclerosis. The injury regresses if the serum cholesterol falls below a certain level. Once the necessary fatty state in serum is established, arteriosclerosis may develop in a matter of days or even hours.

Instead of using thiouracil or thyroidectomy to induce arteriosclerosis in dogs, a laboratory may save time and effort by using radioactive iodine to depress thyroid activity. Curiously enough,

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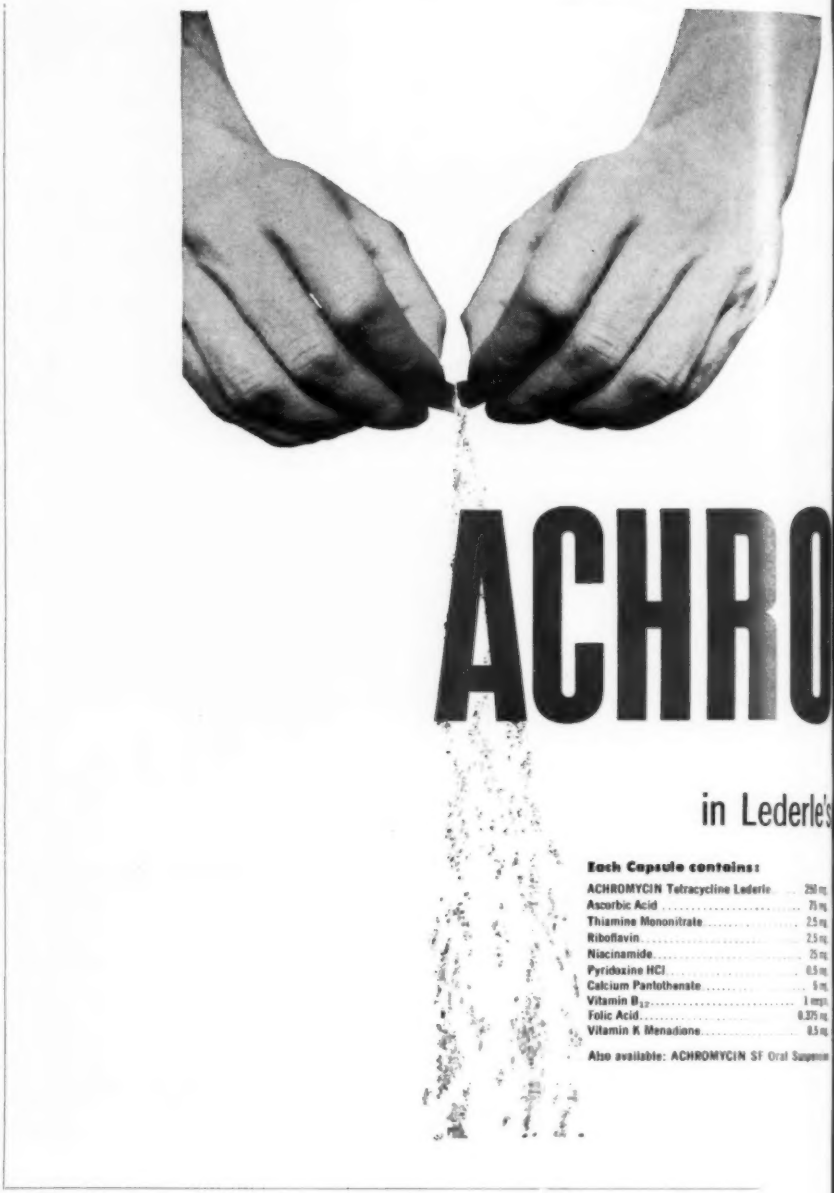
*To summarize in systematic order the reports of progress in arteriosclerosis research supported by grants of the Public Health Service, Dr. Murray Goldstein prepared for the National Advisory Heart Council and for limited distribution a detailed and documented account of the studies and their relation to the pathogenesis, treatment, and prevention of this disease. Dr. Goldstein is an assistant to the chief, Grants and Training Branch, National Heart Institute, Public Health Service.

This paper, reprinted from *Public Health Reports*, March, 1955, was drawn from the original report. It aims to call attention to the general scope of the research, rather than to evaluate individual findings or to describe them in precise detail. At this stage, with so much still unknown and with many studies still in progress, it is not possible to establish an accurate perspective. Also, this paper excludes much valuable work that is unpublished or is supported by institutions other than the Public Health Service. Compressing the information and arranging it in this context has inevitably resulted in somewhat arbitrary statements and has perhaps strained the intended meaning of the reports by the scientists.



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the loss of thyroid activity resulting from surgery, followed by an associated rise of cholesterol in the blood, does not produce coronary atherosclerosis in patients with a history of cor pulmonale, a heart disease secondary to disease of the lungs, or rheumatic heart disease.

A process for inducing atherosclerosis for experimental study, without prolonged cholesterol feeding, consists of planting diethylstilbestrol, a synthetic chemical with action resembling a female sex hormone, under the skin of a chicken. This chemical produces a sustained condition of excess lipid in the serum of chickens and, consequently, arterial lesions which closely resemble those found under controlled conditions.

Rats resist efforts to induce arteriosclerosis even when diabetic, although it is common to find atherosclerosis associated with diabetes in humans. On spectral analysis, the fatty proteins in rat serum differ from those of other experimental animals. Rats, which fail to develop a high level of cholesterol in the blood on a fatty diet, rapidly develop such a condition on the identical diet when rabbit serum is transfused into them. This experience suggests that the difference between rat and rabbit plasma may be the basis for the difference in susceptibility of these species to arteriosclerosis.

A study of the relation between physical injury to the artery wall and the formation of atheromatous plaques finds that weanling albino rats are remarkably resistant to the consequences of injury induced by injection of sodium acetylsulfathiazole. The severity of the consequent lesions increases with the age of the rats injured. Injury to the artery wall of rabbits subsequently fed a high cholesterol diet produces a lesion similar to human arteriosclerosis. Of associated interest, microscopic studies reveal hemorrhage within the artery wall in sudden human deaths from coronary artery disease and coronary atheromatosis.

FUNDAMENTAL STUDIES

The driving forces in the arteriosclerotic process may be in the fluids surrounding the artery wall, in the behavior of the cells of the wall itself, in various forces of environment or custom, in genetic influence, or in a combination of several conditions.

The Artery Wall

Studies of the cellular metabolism of the artery wall itself have been stimulated by the realization that atherosclerosis may occur without the appearance of excess fat in the plasma. One such study demonstrates that the rat artery may convert acetate into fatty acids and can incorporate phosphorus into the phospholipid molecule. The artery wall itself thus can contribute to the production of lipids. To facilitate similar studies, an investigator has developed a technique for measuring the metabolism of intact arterial tissue under aseptic conditions.

Some spontaneous coronary artery lesions of birds are found to be fibrotic rather than fatty; fats may have a late part even in the forming of arteriosclerotic plaques in the aorta of old dogs. Another investigation finds that mucopolysaccharide deposits occur before the lipids. These studies are associated with other efforts to work out the precise cellular history of what appears to be a proliferative and degenerative disease.

The aging factor in the arteries has been associated with the formation of bone or cartilage in the aorta of senile rabbits. Calcification of the media, another sign of aging, is said to precede the formation of intimal plaques, although this conclusion is not supported by other studies. Autopsies demonstrate that arteriosclerosis increases with age in both sexes. It is more severe, however, in the male. The relatively greater susceptibility in boys appears before their 10th year.

Still other techniques are being developed to pursue the study of the role of the arteries in the hardening process.

The Artery's Surroundings

Studies of the artery's chemical and physical environment deal with three main fields: cholesterol and phospholipids; lipoproteins; and physical forces, including blood stream pressure and turbulence.

The presence of cholesterol and its esters in diets, in plasma, and in arterial plaques associated with arteriosclerosis has commanded particular attention.

The work of identifying, measuring, and exploring the metabolic processing of these lipids deals with the production

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and destruction of cholesterol, the differentiation between cholesterol taken in the diet and that formed by the body, and the activity of "trace companions" of cholesterol as atherosclerotic agents.

The fate of cholesterol in the intestine is affected by certain micro-organisms and by chemical conditions which may prevent or accelerate its absorption into the blood stream.

The liver is the chief organ for removing excess cholesterol from the blood. It excretes 60 percent of the excess as bile acid. When unknown forces prevent the return of plasma cholesterol to the liver cells, an excess of fat appears in the blood.

Young animals appear to form more cholesterol in the liver than their elders. Low calorie diets reduce the rate of such formation; high calorie diets restore it. The rate is depressed by a high fat diet, which also seems more effective than a low fat diet in removing cholesterol accumulated in the liver of rats. A high level of cholesterol in the blood does not appear in dogs with deficiency of magnesium or in the liver of rats with a deficiency of pantothenic acid. A deficiency of pyridoxine is accompanied by a high level of cholesterol in the blood of Cebus monkeys and rats, but not in rabbits. Even with a calorie deficiency, chicks on a high cholesterol diet have excess cholesterol in the blood and develop atherosclerosis. As calorie intake rises, successive increments of blood cholesterol decrease. Other dietary fats in combination with cholesterol appear to induce higher fat and cholesterol levels in human blood than cholesterol alone.

Detergents, which have a wetting action that keeps fats and oils in suspension so that they do not cling to affected surfaces, increase all blood lipid components, especially phospholipids. An increase in phospholipids appears to retard or prevent atherosclerosis. The ratio of blood cholesterol to blood phospholipid (C/P ratio) may be of importance in retarding atherosclerosis.

Two substances found in the blood stream, known as lipfanogen and antilipfanogen, respectively, support or oppose the forming of visible fat granules in tissue culture. As cholesterol combines with antilipfanogen, the opportunity for lipfanogen to create fat is enhanced.

Obstruction of the bile duct has been associated with high cholesterol levels in the blood. On the assumption that these levels are supported by the action of retained bile salts, it was discovered that cholate impedes the transfer of cholesterol from plasma to liver. High blood cholesterol may be a development secondary to disorder of cholate metabolism.

In addition to occupation, housing, and dietary patterns, genetic factors may be significant in the tendency to form cholesterol.

Cholesterol and phospholipids in blood exist in combination with proteins rather than in a free state. Such combinations are known as lipoproteins. The amount of lipids in such proteins is measurable by a flotation process which permits analysis according to the density of various classes of lipoproteins in plasma. These are classified in terms of Svedberg flotation (Sf) units. Four laboratories engaged in such investigations are participating in a cooperative study on lipoproteins and atherosclerosis.

Efforts to measure the volume and forms of lipoproteins in the blood and in fixed tissues are proceeding with the prospect that these measurements may help to predict the imminence of atherosclerotic change, to diagnose the present degree of atherosclerosis, and to guide the management of the condition and its consequences. If it is determined what classes of lipoproteins are most formidable, preventive or therapeutic measures may follow.

Because the level of serum cholesterol and lipoprotein has been found to vary in men over a 10-week period or more, single readings may not serve as a reliable indication of a patient's condition.

The liver, one of the organs which produce lipoproteins, increases its output of certain classes if it is chronically inflamed or if it is poisoned by carbon tetrachloride. Hepatectomy causes a gradual decline in all lipoproteins.

Animal species vary in their typical patterns of lipoproteins and in their susceptibility to the effects of different lipoprotein classes. Young women have more

alpha lipoprotein fractions (associated with alpha globulin) and fewer beta fractions than young men. Many men and older women tend to have lower levels of albumin and alpha lipoproteins and relatively higher levels of beta lipoproteins than young women. (This information correlates with the greater tendency of men and older women to develop atherosclerosis.) The same tendency appears in patients with clinical signs of atherosclerosis, as well as among persons with diabetes, nephritis, and xanthomatosis, even when their cholesterol or C/P ratio is not elevated. The level of Sf 12-20 beta lipoproteins is higher in persons with diabetes if they are also arteriosclerotic. The concentration of such lipoproteins has been reported to be a more accurate indicator of atherosclerosis than the cholesterol level. In some circumstances, it is this lipoprotein factor which links obesity with atherosclerosis.

Effects of heparin, a normal trace constituent of the blood, are undergoing extensive study in the search for a chemical which may influence the formation of arteriosclerosis.

Physical forces examined in relation to arteriosclerosis include surface tension (detergent action), filtration of fats by artery and capillary walls, turbulence of blood in the aorta of rabbits, and elevated blood pressure (hypertension). Although hypertensive patients show a high incidence of arteriosclerosis, the experimental evidence so far does not establish that the one condition necessarily induces the other.

CLINICAL STUDIES

There have been no research projects on the incidence of morbidity from arteriosclerosis because of the difficulties of reliable diagnosis.

A reliable method by which the incidence or severity of arteriosclerosis could be measured objectively has been the goal of many investigations. Determinations of serum cholesterol, cholesterol tolerance, lipoprotein fractions, phospholipids, cholesterol-phospholipid ratio, lipfanogen-antilipfanogen ratio, or neutral fat have been suggested as possible indexes of the probability of developing or having already developed arteriosclerosis.

In man, excluding histological study after biopsy or necropsy, the only reliable method of diagnosis at the present time depends on the onset of symptoms associated with a decreased blood supply to specific organs or parts of the body. For better objective evaluation of these observations, many methods and devices have been suggested and are being perfected; some of these are flicker flame photometry, plethysmography, ergometry, electrocardiography, vector cardiography, ballistocardiography, vascular catheterization, roentgenography, and angiography. As yet, no generally acceptable means has been agreed upon for the preclinical diagnosis of arteriosclerosis.

PROPHYLAXIS AND THERAPY

Diet, medication, surgery, and rehabilitation are being studied as means of preventing or treating arteriosclerosis.

The administration of adrenal cortical and corticotrophic hormones is associated with high levels of cholesterol and lipids in the serum without an increase in lipoproteins or in atherosclerosis.

The relative susceptibility of males to arteriosclerosis has encouraged studies of the effects of synthetic and natural sex hormones on the occurrence and treatment of this disease.

Studies of thyroid effects find that administration of thiouracil or thyroidectomy does not cause regression of arteriosclerotic lesions in dogs.

After biological experiments indicated that a portion of the mammalian brain affects the movement of cholesterol into the blood stream, a lipid-poor, cholesterol-free residue of this organ was fed to patients with arteriosclerotic heart disease and a high level of cholesterol in the blood. Their serum cholesterol fell "significantly," presumably as a result of the chemical effect blocking the absorption of sterols in the intestine.

Common "lipotropic agents" necessary for fat metabolism, such as choline, methionine, and inositol, have no beneficial effects on the incidence or severity of arteriosclerosis. Ferric chloride decreases absorption of cholesterol from the alimentary canal but not its production by the liver. Other chemicals which reduce absorption are sitosterol and dihydrocholesterol.

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1. Lobitz, W. C., Jr., and Jillson, O. F.: Postgrad. Med. 12:2, 1952.
2. Goodman, H.: J.A.M.A. 129:707, 1945.
3. Underwood, G. B., et al.: J.A.M.A., 130:240, 1946.
4. Lubowe, I. I.: New York State J. Med. 50:1743, 1950.
5. Nomland, R.: Postgrad. Med. 11:412, 1952.

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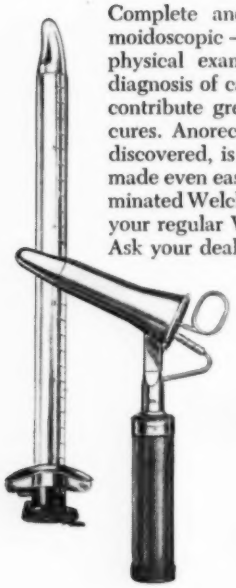

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The prospect of prophylaxis by diet is not materially brightened by studies to date. A decline in Sf 12-100 lipoproteins accompanies a loss of weight. The Kempner rice diet reduces serum cholesterol and the C/P ratio. It is not certain that restriction of cholesterol in the diet will ordinarily reach the level needed to produce clinical results. The amount of cholesterol in the diet may vary widely without producing marked effects on the levels in the blood. Restriction of cholesterol in the diet must be accompanied by considerations of total nutritional needs, such as the part proteins play in lipid metabolism.

Surgery to repair or replace vessels damaged by arteriosclerosis has employed a variety of techniques. These include generation of intercoronary vessels in a heart inflamed by abrasive asbestos powder; grafting of substitute arteries which have been dehydrated or taken out of cold storage; the use of prosthetics made of metal, plastic, latex, or thorotrast; and the patching of arterial walls with peritoneum. The technique of grafting has evoked studies of optimal length, source, structure, sterilization, and storage of blood vessels.

Studies of rehabilitation of cardiac patients are actively encouraged. Energy requirements, as measured by oxygen consumption, are being measured for various occupational activities.

Most of the clinical studies noted here are undertaken in association with experimental laboratory studies, in order to assure that the patient will have the advantage of validated theories, technical experience, and full understanding of the biochemical factors.

THE MARCH OF MEDICAL SCIENCE*

C. J. Van Slyke, M.D.†

I understand that this series of meetings seeks a hard, realistic appraisal of our health future—what tomorrow's developments may be, what problems these developments may engender, and what we need to do now in order to be prepared for what tomorrow may bring. I believe that at one point this forum was labeled by its sponsors as "an experiment in practical forecasting"—an intriguing phrase, indeed.

Our part of the program this afternoon is "The March of Medical and Social Sciences." This title in itself reflects one of the significant trends of the times—the growing awareness that health is an inseparable element in social progress, and that medical science does not and cannot function in isolation from the social, economic, educational, cultural, and other forces in our society.

RESEARCH ADVANCES

The problems faced by medical science are tremendous in volume, scope, and complexity. When one looks back at what medical science has accomplished over a span of several decades, he can identify the items quite easily. When one looks at medical science today and tries to pick out the most promising work, he finds the task quite difficult. But when one attempts to anticipate what progress will have been made by, say 1975, his first inclination is to look for cover, and his second is to generalize and digress. For the record shows that medical progress is replete with false hopes, pitfalls, and hypotheses that cannot be confirmed. The way to progress is long and difficult, and the specific advances are anticipated only by those who possess temerity and clairvoyance.

It is possible, however, to pick out several broad general areas in which progress is being made today and to project them ahead for several years, as-

*Delivered at the panel on "The March of Medical and Social Sciences" March 23 at the 1955 National Health Forum on "Forecasting America's Health," Sheraton-Astor Hotel, New York City, sponsored by the forty-nine national organizations working together in the National Health Council as part of the Council's 35th Annual Meeting.

†Associate Director, National Institutes of Health; Assistant Surgeon General, U. S. Public Health Service.

suming that no new variables are brought into play. I do this as one whose principal orientation has been to laboratory and clinical research and to the hospital environment. I cannot go into fields that are foreign to my experience, and I will try to confine my speculations to areas which might have meaning in terms of planning health resources for the future.

It is reasonable to assume that there will be vaccines which will be largely effective in the prevention of influenza, poliomyelitis, and the minor upper respiratory infections, including the common cold.

There could be major advances against heart disease, featuring the prevention of rheumatic heart disease by the prevention of rheumatic fever, drugs to control hypertension, and some as yet unknown means to control the fatty deposits in the blood stream which lead to atherosclerosis.

There will be improvements in cancer diagnosis. Treatment of cancer will improve, too; in addition to x-ray therapy and surgery, there may well be chemical agents of particular value against the soft tumors.

There will be new drugs useful in the management of mental illness. We will have better knowledge of prevention. But the hard core of the problem of mental illness will remain until training and education begin to yield the professional staffs that are so desperately needed, both to study mental illness and to care for the mentally ill.

There will be a series of new drugs, both natural and synthetic, useful in the treatment of arthritis and other rheumatic diseases.

We will have the knowledge and methods to prevent several of the leading forms of blindness, particularly among children.

There may be fewer advances in the management of neurological disorders, particularly multiple sclerosis, cerebral palsy, and muscular dystrophy. However, new rehabilitation techniques are in process which bring some of the victims of these diseases a measure of independent self-care.

These items of progress and future accomplishment are typical. One could go on and on with such an enumeration, getting more and more specific. In total, progress such as this is encouraging and at the same time challenging. It offers the hope that through sustained research on a broad front, we can gradually find some of the key answers that are needed, in order to reduce a portion of the burden and suffering of disease, disability, and premature death.

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THE MEDICAL SCIENCE SPECTRUM

As one studies the pattern of research activities in the medical and biological sciences, it becomes apparent that while a great deal of progress is being made toward relief or amelioration of the effects of diseases, we are not making the same progress toward understanding of the basic underlying causes of disease. This is particularly true for the chronic illnesses. I raise this point only to emphasize the continuing need for fundamental studies which will yield the knowledge with which we can achieve the ultimate goal—prevention.

By these fundamental studies, I am referring, in part, to the clinical and laboratory investigations which are traditionally thought to be the way to establish causation. But I am also referring to the opposite end of the research spectrum—to those studies involving man in his environment, both normal and abnormal. If medical science applies the knowledge and techniques of epidemiology, statistics, economics, sociology, anthropology, and related fields to these fundamental investigations, it may find that such fields have as much potential for identifying the factors in disease causation as the clinical and laboratory studies.

For medical science has concentrated on understanding the nature and modes of action and interaction of progressively smaller things. Not too many decades ago, we were studying functions of whole organs or large sections of tissue. Gradually, we reduced the dimension of our studies to the cellular level. Now we are concerned with subcellular matters. We find that even within the cell, there are relations and interdependencies which materially affect the life processes. Scientists study, for example, the role of cytoplasm in genetics, the ability of viruses to destroy certain cells but live

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1. *Cass & Wolf: Gastroenterology 20:149, 1952*
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peacefully in others, or the factors that govern cell growth and multiplication. They are prone to use the molecule as the smallest unit of a substance, yet we know from the mushroom-shaped clouds over Yucca Flats that even the elements that compose the molecules are not the ultimate in reduction.

I do not, of course, challenge the validity of the scientific method which seeks understanding of the processes of health and disease through this microscopic and submicroscopic approach. I stress, rather, that it is time for us to also give emphasis to the study of man as a "supramacroscopic" organism, if you will—and to study the whole man in relation to other men and to the environment in which they live.

By way of illustration, we are now conducting a study of a neurological disease known as amyotropic lateral sclerosis, more commonly called "Lou Gehrig's disease." It has been found that on the island of Guam, this disease is fifty times more prevalent than it is in the United States. The situation offers a golden opportunity for an epidemiological study. Or there is another study in Framingham, Massachusetts, which has undertaken to follow a large population group in order to investigate and evaluate the factors involved in the development of hypertension and atherosclerosis. For this kind of study, the investigators are not only the physicians, but also the epidemiologists, the biostatisticians, the social workers, the nurses, the public health workers, the industrial physicians, the sociologists and psychologists, the anthropologists and historians—all those who can contribute to better understanding of man and his emotional, intellectual, and physiological responses to the world about him.

In a sense, there are many similarities between the single cell and the whole man, when each is studied in relation to its environment.

FORMULA FOR PROGRESS IN MEDICAL SCIENCE

Progress in medical science does not come easily. But I am convinced that it will continue in the future as it has in the past, and perhaps more abundantly. We have only to build intelligently on our experience and take the time to shape our efforts toward the central problems and the major goals.

There is a simple—a deceptively simple—formula for such progress. Start with trained people (who, because they are trained, have sound ideas of their own about how a problem can best be attacked). Add facilities (which means not only buildings, equipment and supporting services, but also a creative environment with challenging interplay among the professional staff). Then add money (a necessary commodity, even in these fields which are characterized more by self-sacrifice than by self-service). The result: progress.

This formula can be readily visualized in the form of a three-legged stool; one leg is money, one leg is facilities, and one leg is manpower. It is clear to me that we have a wobbly stool, because the manpower leg is shorter and weaker than the others. The most serious threat to the ability of medical science to do the job it must do is the impending shortage of trained investigators of all kinds. Science teaching in the secondary schools has not kept pace with increasing needs. The preclinical sciences have suffered in the colleges and universities. And there are too few career opportunities in the basic sciences related to medicine and biology.

We must do something immediately and constructively to resolve this issue, lest the stool on which our health structure rests might collapse because of this imbalance. All of us in the health field have an obligation to assist in the resolution of this problem.

COOPERATIVE EFFORT

Another challenge of medical science is to achieve a more effective linking of institutions and agencies and organizations which together make up the health resources of our Nation.

I do not mean to over-sell the value of coordination as such. There are times when I feel we spend so much time coordinating our efforts that the efforts themselves get almost obliterated, and coordination becomes merely entanglement. Cooperation, yes—knowledge of other programs, ability to use them as resources, eagerness to work together where interests merge, respect for the objectives and methods of other groups, desire to avoid jurisdictional disputes—

these are the essence of effective cooperation in health and related fields.

We need more of this kind of cooperation. For as we get larger and older as a Nation, the health needs of the people grow and the pattern of health services changes to meet these needs. In this kind of dynamic situation, we should not lose sight of the fact that we—all of us—have but one ultimate goal for our efforts (health for the individual) and but one way to achieve this goal (by improving and extending our health resources, so that they provide more services for more people).

THE PHYSICIAN AND THE NATIONAL HEALTH SCENE

I do not need to detail for this audience what we mean by America's health resources or to describe the methods by which the component parts work. The hub of the wheel, of course, is the physician. Most of the health resources relate to his work.

Not too long ago, medical practice by the physician, together with nursing and pharmacology, was virtually our only mechanism for coping with disease. The individual physician—with his portable knowledge, his bag of simple instruments and drugs, and his art and common sense—was practically the whole institution of medicine.

Today the physicians, far more effective than their early predecessors, are still the spearhead of the medical force. But their effectiveness is augmented by an army of allied workers that back them up. Whether general practitioner or specialist, the physician depends on the resources of the pharmaceutical industry, the modern hospital and health center, the analytical laboratory, and complex precision apparatus. His knowledge and skill are multiplied by organization and technology.

The physician is also buttressed by voluntary health agencies, State and local health activities, specialized medical care institutions, specialists in rehabilitation techniques, and by the fruits of research. Thus has the jigsaw puzzle of modern medical science taken shape. If we are going to keep on using it, it is essential that no piece be lost or damaged.

THE GENERAL OUTLOOK

Probably the largest single factor to concern those who plan civilian health programs in this country is our changing population. In all of the more civilized countries, populations are growing—both larger and older. With the progress of medical science, particularly against the fatal infections of childhood, the average life span has progressively lengthened. More people are reaching maturity and old age. We can surely predict that the principal medical problems of the second half of this century will be those deriving from population density and the number of older people in our midst.

Specifically, the diseases that have come sharply into prominence during the last decade—heart disease, cancer, mental and neurological disorders, arthritis and various metabolic problems, blindness and dental disease—will reach even greater proportions. These are the chronic diseases, responsible even now for a vast army of victims with varying restrictions on their productivity. The future will bring increased pressures upon society to prevent or cure these diseases—to prolong the usefulness of man throughout the maximum portion of his allotted years.

This seems to me a herculean task, but not an impossible one. At a growing rate, we have seen new tools and techniques emerge from the laboratory of the research scientists and find application in the hands of physicians and allied health workers. Every day, new facts are added to the stockpile of knowledge—often so basic that the scientist himself can visualize no immediate application, but from which the scientists of tomorrow can fashion new diagnostic tools, new preventive vaccines, new drugs for treatment.

The emphasis on chronic diseases should not obscure the fact that serious infectious diseases are with us, too—hepatitis, influenza, brucellosis—and that any relaxation of public health vigilance is an invitation for reappearance of smallpox, typhoid, and other scourges of the past. We must continue to find answers to these questions, too; and the search becomes all the more meaningful when one remembers that some of the major chronic diseases may well have

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their origin in microorganisms. Witness, for example, the several types of cancer in animals which are known to be caused by viruses.

Another problem for the future rests on the fact that this country has become predominantly urban. This trend increases the burden of State and local health departments to maintain sanitary, healthful conditions for our 160 millions. It brings to the fore such programs as city health planning, air and water pollution, accident prevention, sanitation, and cancer that may be of industrial origin.

I think I should make special mention of two problems of major significance—mental illness, and diseases of aging. Already the mentally ill occupy half of America's hospital beds and cost us well over a billion dollars in tax money alone. And the dimension of the problem is growing all the time. We must certainly find better preventive methods and more practical and adequate means of treating these unfortunates who are incapable of functioning effectively in our increasingly complex society.

Related problems are juvenile delinquency, drug and alcohol addiction, the gamut of self-injurious and antisocial behavior. These are stubborn problems that must be approached through both the physical and the social sciences, as well as through legal and other means.

Gerontology, the study of the processes and effects of aging, assumes its true importance when one realizes that by 1975, over 20 million people in this country will be more than 65 years old. The problems posed by this simple fact extend across the full range of the health sciences. We need to know the physiological, biochemical, and psychological changes that take place in aging man. Assuming a reasonable degree of success in research on chronic illness, and some new understanding of the aging process through gerontological research—what then? Inevitably, the average life span increases some more. It was 27 years in the time of the Romans, 47 in this country in 1900, and is almost 70 in this country today. There are some good people who forecast 100 years as the average life span!

So we return to my earlier reference on the inseparability of medical and social science. It is all too obvious that we must do something about effective utilization of our older people, reexamining our whole social and economic structure to find ways to help them recapture their place as useful and productive and happy members of society.

We must keep our feet on the ground, however, with regard to the potentiality of medical science. We will not find the ideal solution to all or even most of our health problems within the next 25 to 50 years. Great strides will be made. But we can never forget that the sick and the disabled will always be very much with us—that we will be faced, for many years to come, with the need to provide better care for the ill, better use of the treatments at hand, and more intensive efforts toward the prevention of disease. For all of these, the improvement and extension of health facilities and personnel is mandatory, so that we can cope more adequately with the problems of today and be better able to meet those of tomorrow.

CONCLUSION

In summary, then, these points seem to me to be most germane to the forward march of medical science:

1. The great progress that has been made in the last half-century has been largely against the infectious diseases.

2. The great problems of the next half-century will be those associated with our aging and expanding population.

3. A successful attack on chronic and environmental illness demands both an expansion and a reorientation of our total health resources—both medical and institutional care, public health practice, medical and public health research, teaching and training programs, health education, and community and voluntary agency activities.

4. We must move simultaneously along three lines: toward better application of what we now know about the prevention and control of disease; toward the acquisition of new knowledge; and toward making the necessary changes in today's health structure so we will be ready for tomorrow's challenges.

An occasion such as this National Health Forum, bringing together so many

people with different training and representing different organizations, but with common purpose, is another demonstration that we are ready to meet these challenges realistically and cooperatively, thus ensuring success.

THE CHEMICAL CONTROL OF CANCER*

By James F. Holland, M.D.†

(Continued from May JOURNAL)

There is no interrelation of sensitivity or refractoriness to cortisone, 6-mercaptopurine, or a-methopterin. A particular child may enjoy hematologic remission from any, all, or none of these drugs (*36*). No criteria are yet available to predict susceptibility other than clinical trial; accordingly, consideration must be given to clinical management and the choice of antileukemic drug which is to be used first. When the severity of the patient's disease threatens imminent demise or catastrophe, cortisone is best employed, because of its rapidity of action and value in thrombocytopenic hemorrhage, in an attempt to assure survival for a trial of the other drugs. Of the antimetabolites, it would seem safer to begin with methotrexate because folinic acid has at least the potentiality of competing with methotrexate in the event serious drug toxicity occurs.

Uremia, attributable to uric acid crystallization within the kidney, has been described following the use of several different chemotherapeutic agents in instances where extremely rapid tumor destruction ensues. This is a rare complication of acute leukemia when a precipitous fall in leukocytes follows institution of therapy.

Cortisone administration produces considerably less objective evidence of improvement in acute leukemia of adults than of children. Its administration is often attended by some symptomatic benefit, however, which makes it worth while. The lymphocytic leukemias, in chronic or subacute phase, may respond favorably with decrease in leukemic cellular infiltrations and increase in normal marrow function. In a small proportion of patients with Hodgkin's disease or lymphosarcoma, objective diminution in tumor tissue can be demonstrated, and in a larger group, clinical improvement occurs without appreciable change in node masses (*41, 42*).

Cortisone often stops bleeding due to thrombocytopenia without increasing the platelet count or modifying the cause of disease in any other way. Decrease in hemolysis associated with splenomegaly in the lymphomas and leukemias may be detected during cortisone administration. Most of these effects are of short duration, but the important temporization afforded frequently allows return of marrow function before a myelotoxic insult is again sustained. Conversely, there is no apparent effect of cortisone on the pancytopenia caused by those drugs toxic to marrow function which are described in this review.

Recently, the use of large doses of cortisone (0.3 gm. daily) in treating metastatic carcinoma of the breast has been reported to produce objective evidence of regression in nearly half the patients (*43, 44*). Favorable response, where observed, was unrelated to ovarian or adrenal function, since prior surgical removal of these glands did not influence the results. The longest objective remission from cortisone lasted 3 months; symptomatic improvement also occurred briefly in another third of the patients. At lower doses of cortisone (0.1 gm. daily or less) or with moderate doses of corticotrophin, some subjective improvement, but no objective regression has been seen (*45*).

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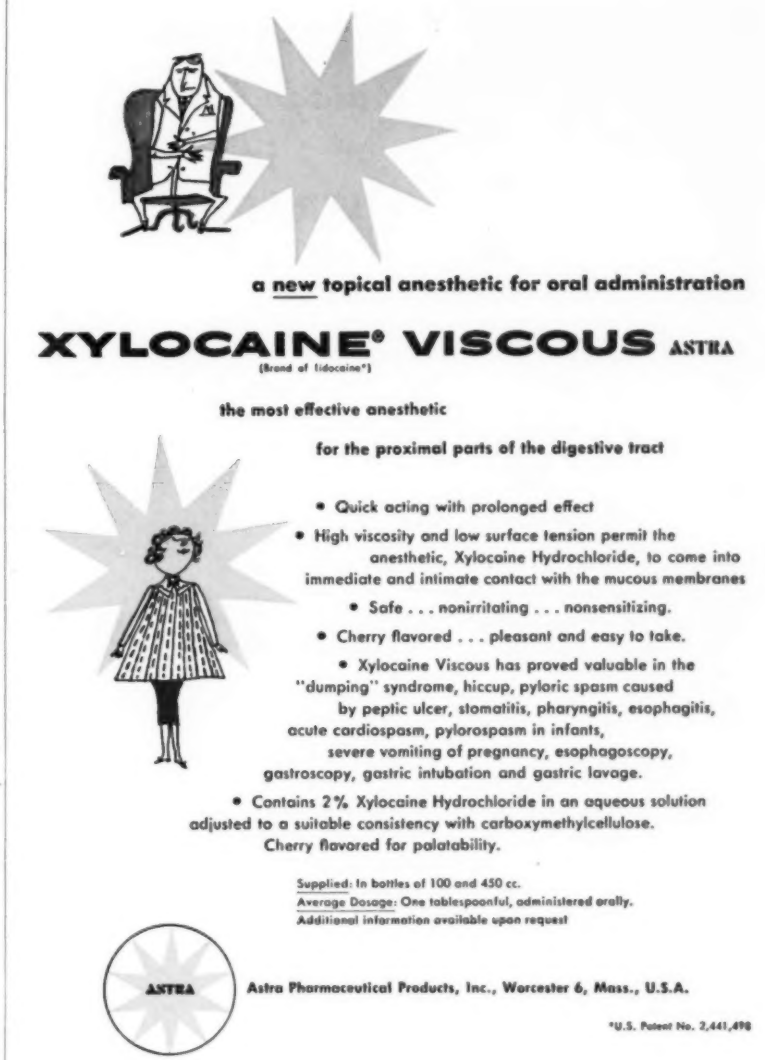
*Reprinted from *Public Health Reports*, December, 1954.

†Dr. Holland, previously with the National Cancer Institute, Public Health Service, is now associate chief of medicine at Roswell Park Memorial Institute, Buffalo, N. Y.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metastatic carcinoma of the breast. A feeling of well-being is experienced by nearly two-thirds of the patients, and oftentimes dramatic disappearance of bone pain allows ambulation in otherwise bedridden women.

Symptomatic benefits far outstrip the objective responses which can be detected by clinical criteria or even by metabolic balance studies. In only 11-19 percent does regression of osseus lesions take place. Effects on soft tissue metastases occur in about 20 percent of cases but are less dramatic clinically than the response of bone disease; pulmonary, cerebral, and hepatic spread are infrequently improved to a significant extent by the administration of androgens (*46*).

The action mechanism of androgenic substances in metastatic breast cancer is unknown. Since clinical improvement is often observed without delay in the continuing growth and spread of the tumor, it is assumed that most of the beneficial effects derive from influences on the host tissues and the host's response to the tumor rather than from effects on the cancer cells per se. The palliation which results from androgen administration is relatively short-lived (*47*). In one study, of those patients who responded favorably to testosterone, 95 percent did so within 2 months. Reactivation of symptomatology occurred within 6 months in 58 percent and in less than a year in 95 percent (*48*).

The most widely accepted dosage regimen for the administration of testosterone propionate is 0.1 gm. intramuscularly three times weekly. Larger doses of this compound have been found to offer little except an increased intensity of undesired effects (*46*). These phenomena include voice lowering, hirsutism, acne, edema, flushing, and increase of libido (*49*).

Methyltestosterone can accomplish virtually all the actions of testosterone propionate and is a useful oral therapy (*50*). Chemical hepatitis (*51*), which may occur from methyltestosterone treatment, is an uncommon toxicity that is not prohibitive. Stanolone, a new analog, offers no advantages over testosterone propionate (*52*).

In the presence of osteolytic lesions, any of the androgenic compounds can induce hypercalcemia, presumably through stimulation of the local demineralization process with production of a calcium load in excess of the renal capacity for excretion. Hypercalcemia may be manifested by anorexia, nausea, vomiting, constipation, oliguria, azotemia, apathy, coma, and death. The occurrence of hypercalcemia in approximately 10 percent of the patients treated with androgens must be anticipated (*53, 54*). No thoroughly satisfactory treatment for hypercalcemia has been advanced (*55*).

DIETHYLSTILBESTROL

Diethylstilbestrol and the naturally occurring estrogenic compounds are used with good response in the majority of patients with carcinoma of the prostate, and in carcinoma of the breast of males and postmenopausal females.

In males, stilbestrol administration is often a part and prolongation of "androgen control" therapy which includes initial castration. In elderly females, it is said that estrogen administration changes the hormonal milieu to one alien to the cancer cells. Whether, indeed, stilbestrol has pharmacological antineoplastic actions which are independent of its estrogenic properties is not known. Regression of the primary tumor and soft tissue metastases as a result of stilbestrol therapy in carcinoma of the breast in females may be a dramatic event.

In one study, 90 percent of those patients who responded favorably did so within 2 months. The duration of response produced by estrogens was longer than that from testosterone; 30 percent of the patients were still in remission after 12 months, and 6 percent had not relapsed after 2 years (*48*). Degeneration of tumor cells and cicatrization of the tumor area, far in excess of similar phenomena in untreated cancers, has been demonstrated (*56*).

The association of significant benefit from estrogen administration and postmenopausal status of at least 5 years, without regard to chronologic age, is persuasive. Major tumor regression has been produced in 24 of 99 women 5 years or more postmenopausal, whereas, only 1 of 42 patients below these limits sustained similar regression. Eight of the 42 younger women suffered exacerbation of their cancer from estrogen (*48*). The use of estrogens in this latter group offers considerably less than androgen

therapy. For the more advanced postmenopausal patients, however, stilbestrol is the most satisfactory and practicable drug (*46*).

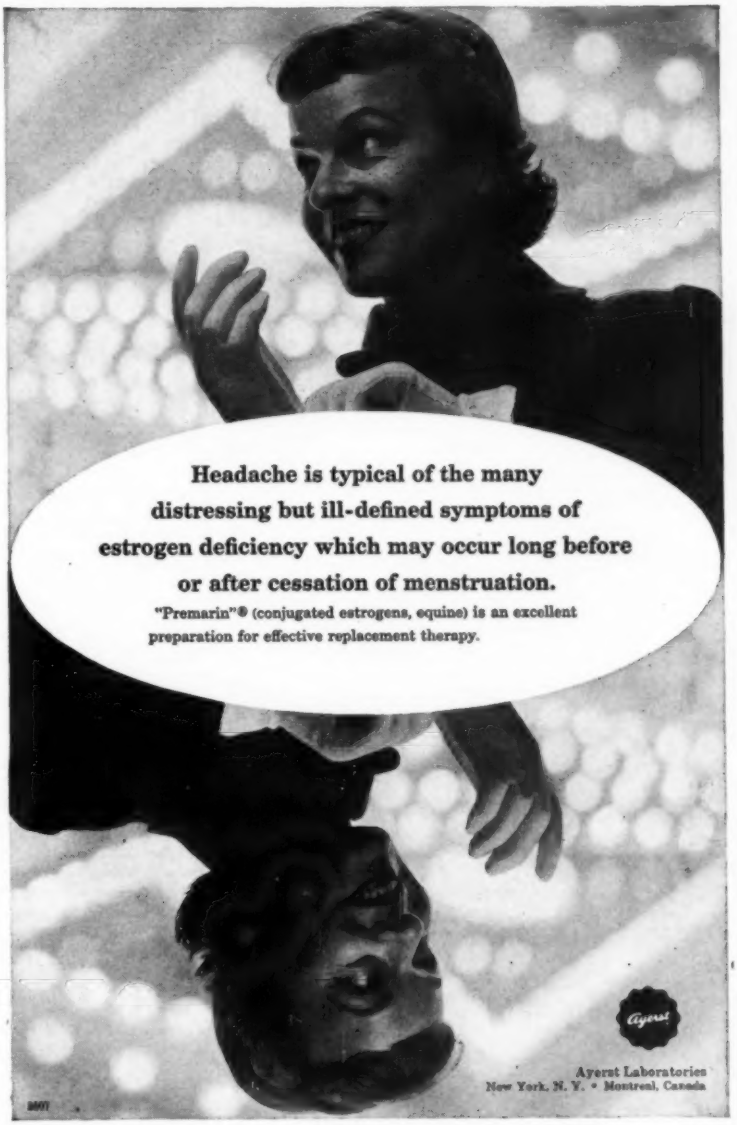
The frequency of osseus metastases in older women is less than that in the physiologically younger age groups where testosterone is usually employed. In these more aged patients, however, equally good regressions of bone metastases are effected by stilbestrol. Nearly one-half of properly selected patients have regression in breast, skin, and lymph node spread, and one-third experience improvement in pulmonary lesions. Other visceral metastases are less responsive (*46*).

The medical management of prostatic cancer, by hormonal means, is more satisfactory than that for any other disseminated carcinoma. Stilbestrol in small doses is capable of prolonging life and palliating symptoms almost as effectively as castration. Extensive data indicate that a combination of castration and stilbestrol administration reduces mortality and morbidity for prostatic cancer more effectively than either method alone (*57*). Decrease in tumor activity evidenced by relief from pain, palpable diminution in, or disappearance of, tumor masses, reduction in, or cessation of, osteoplastic activity of metastases, and a fall in circulating acid phosphatase are among the benefits seen. Restitution to nearly normal health occurs while the tumor is regressive or static. A marked decrease in the extent and cellularity of malignant tissue is noted microscopically, and diagnostic characteristics of carcinoma may be nearly lacking.

The duration of such a remission in patients who have been castrated and given estrogen therapy is appreciable. Thirty-five percent of men with metastases, and 50 percent of those without metastases, at the time of institution of treatment, survive for 3 years (*65*). Eventually, refractoriness to therapy and exacerbation of the disease occur despite administration of estrogen. A possible explanation of this phenomenon could be the tumor's sensitivity to androgens produced by the adrenal cortex (*58*). Temporary benefit in a fraction of the men at this stage of the disease following bilateral adrenalectomy lends some credence to this hypothesis. The lack of objective response in the majority of patients (*59, 60*), however, suggests that adrenal androgenic stimulation is not the essential support for progressive growth. Rather, a state of relative tumor autonomy, without regard to hormonal support or suppression, seems to succeed the condition of the previously dependent cancer in most patients (*7*). Accordingly, adrenalectomy for prostatic cancer has largely been abandoned.

The response of metastatic carcinoma of the male breast to castration, with or without estrogen, is qualitatively similar to the response of prostatic cancer. The frequency, completeness, and duration of objective and subjective response in the few cases reported (*61, 62*) are better than the response of carcinoma of the female breast to oophorectomy or to hormonal therapy.

Many dosage regimens have been advocated for stilbestrol administration in cancer patients. These vary in the main from 1 to 1,000 mg. daily. A considerable experience has been obtained in the lower dosage range from 1 to 15 mg. per day in divided doses orally. No convincing evidence exists that larger doses accomplish qualitatively or quantitatively different results. The more common undesired effects of stilbestrol are anorexia, nausea and vomiting, fluid retention, breast engorgment (gynecomastia in males), nipple and axillary pigmentation, frequency and incontinence, and interval and withdrawal uterine bleeding. Hypercalcemia while taking stilbestrol has been reported in women with osteolytic metastases from breast cancer (*46, 49*).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in negative calcium balance in patients with metastatic osteolytic lesions subjected to a challenge course of estrogens, oophorectomy and subsequent adrenalectomy have offered benefit, presumably by removal of estrogen-producing tissues (*43*). The position that adrenalectomy will have in the management of metastatic breast cancer is not yet defined. In the meantime, the extent of the procedure, with the hospital and economic burden entailed, properly limits its indiscriminate empiric use.

Hypophysectomy has been performed by one group on nine patients with carcinoma of the breast. In only a single patient is sufficient data available to assess the response, and she enjoyed moderate benefit (*65*). More recently, objective diminution in osteolysis during the course of metastatic breast cancer has been reported following hypophysectomy in two patients who previously had had oophorectomy and adrenalectomy (*66*). The subsequent administration of somatotropin was associated with metabolic evidence of progression of the disease, reflecting a direct effect of somatotropin on human breast cancer. Other reports of failure to improve patients with malignant melanoma (*67*) and adrenal cortical carcinoma (*68*), together with the magnitude of the procedure (*65*), suggest a reserved and critical evaluation of this operation before it is enthusiastically accepted into surgical endocrine therapy.

URETHANE

Urethane is a simple chemical compound first reported in 1946 to be of use in treating the chronic leukemias (*69*). A considerable experience in these diseases has established a place for urethane as a satisfactory alternative to radiotherapy. Continuous administration at levels of 1 to 5 gm. daily may achieve satisfactory response although there is rather wide variation in the dose capable of initiating remission. In 13 patients with chronic myelocytic leukemia, 19 to 134 gm. were required in 11 to 36 days to effect a drop in leukocyte counts to levels approximating 20,000. In chronic lymphocytic leukemia, where response was less regular, even more discrepant results were encountered (*69*). The compound demonstrates no clinical usefulness in the acute leukemias. Anorexia, nausea, and vomiting are prominent side effects, but they may be averted by rectal administration (*65, 66*). Pancytopenia may result from excessive dosage.

The effects of urethane in multiple myeloma are variable. Diminution in plasma cells and myeloma cells in the marrow, decrease in Bence-Jones proteinuria and hyperglobulinemia, relief from bone pain with occasional recalcification, and striking clinical improvement have been described (*71-73*). Such amelioration is inconstant, however, occurring in about one-third of the patients with myeloma, and usually persisting for only weeks to months. Radiotherapy directed to painful osseous lesions frequently affords palliation and is a helpful adjunct. The rarity of objective remission from 6-mercaptopurine (*74*) does not support the routine use of this compound in patients with myeloma. Substantial improvement does occur in a fraction of patients treated with ACTH or cortisone (*75*), and these drugs, because of their relative freedom from serious toxicity, seem worthy of trial in a given patient.

MYLERAN

Myleran is the most active member of a series of aliphatic disulfonic acid esters. This series, because of its chemical resemblance to the nitrogen mustards, was purposefully synthesized in search of compounds with similar biological properties (*76*).

Clinically, myleran has found satisfactory application in chronic myelocytic leukemia (*77,78*) where fall in leukocyte counts, particularly of immature granulocytes, decrease in splenic size, and rise in hemoglobin follow administration. Lymphocytes are relatively resistant to the action of the drug, but decrease in platelets occurs in virtually all patients and the decrease may continue for many weeks after initiation of treatment. Bleeding may supervene when thrombocytopenia is sufficiently marked.

Wide variation in dosage regimens has been studied. The most satisfactory would appear to be maintenance therapy, at about 4 mg. daily, with or without an initial loading dose, in patients checked frequently with blood counts. Under such a treatment regimen, approximately half of the patients with chronic myelocytic

leukemia whose treatment has been reported have enjoyed clinical remission and hematological control for 6 months or longer.

Resistance to therapy has been encountered in some patients after initial response. Although patients believed to be refractory to further radiotherapy have responded, in certain instances dramatically, remissions have been rather unsatisfactory and brief when the cell type has been characterized by a higher proportion of myeloblasts, as is frequently the case in the preterminal radioresistant stages of the disease. Acute leukemia has not been benefited (*77, 78*). Although myleran is effective in suppressing many rodent neoplasms, it has been found ineffective in a variety of metastatic human carcinomas (*79*).

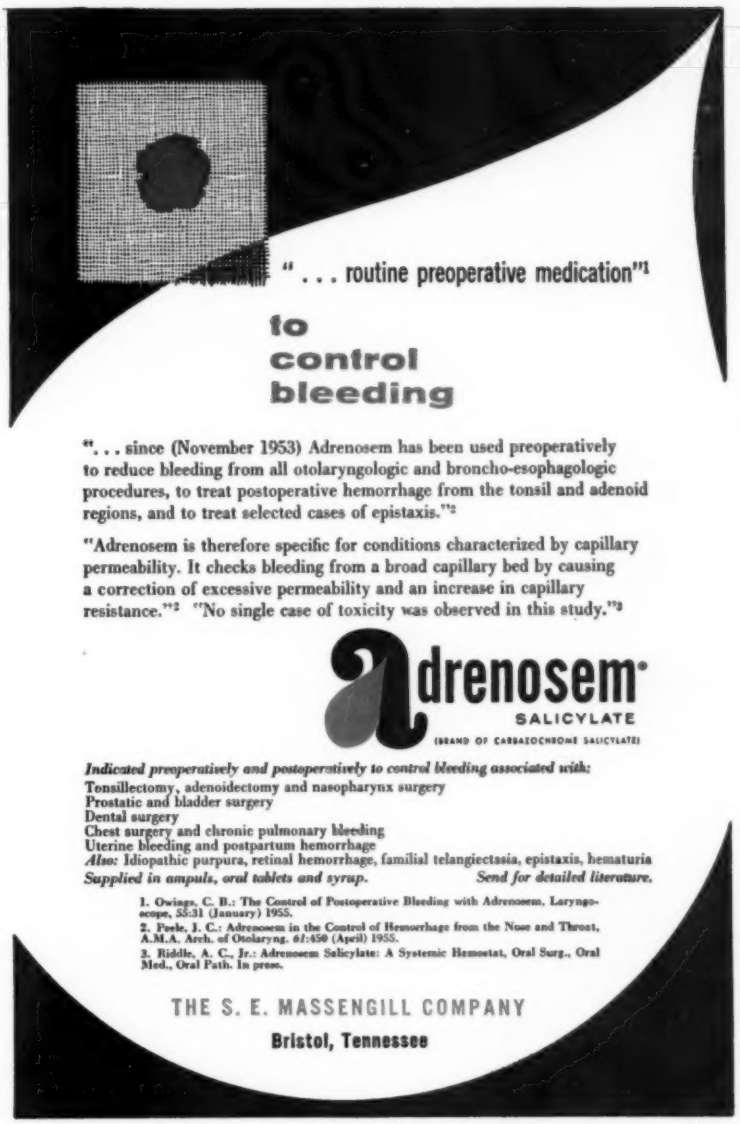
ETHYLENE PHOSPHORAMIDES

The actions of mechlorethamine and triethylene melamine in experimental and clinical cancer have stimulated the synthesis and investigation of other compounds which carry ethylenimine groups. Two of the ethylene phosphoramides, triethylenephosphoramide (TEPA) and triethylenethiophosphoramide (thioTEPA) have been subjected to clinical trial.

In rats, TEPA and thioTEPA have a spectrum of anticancer effects similar to one another and to TEM (*80*). In patients with a variety of malignant neoplasms, the results following TEPA administration have approximated those with TEM (*81, 83*). It has not achieved wide clinical application.

ThioTEPA has produced satisfactory initial responses in patients with chronic myelocytic leukemia (*84*). Regressions of metastatic carcinomas of the breast and ovary, and of other miscellaneous sites have been reported to occur with considerable regularity following its use by parenteral injection (*84, 85*). Particularly satisfactory remissions have been described after injection directly into a tumor mass.

Inadequate time has elapsed for critical appraisal of this compound by other investigators; the passage of time will also permit evaluation of the duration and clinical value of the response elicited so far. It seems doubtful that intratumor injections should be required if the drug has striking anticancer effects.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CONCLUSION

The chemical control of cancer has not yet been realized. Within the past 15 years a more profound understanding of cancer has been achieved, and several approaches to its treatment with drugs have been reviewed in this paper. To an appreciable extent, the advances which have been made stem from knowledge and intent rather than from happenstance.

Cancer cells are different from their normal counterparts when viewed under a microscope. Although it is possible that this difference is purely one of rearrangement of the same chemical components within the cell, a vast amount of evidence defines at least quantitative alterations in composition. Functional capacities of malignant and normal cells of homologous tissues may be greatly different. Therapeutic agents which depend on these quantitative differences in the metabolism of cellular constituents afford promise for future progress. A constant search for qualitative differences between neoplastic and normal cells may elucidate obvious pathogenic or perpetuating mechanisms which can be interrupted. Further, it seems within the realm of possibility that pharmacological agents will be found that can effectively counteract the etiological factors which lead to cancer—be they viral, chemical, or physical exposure; idiopathic mutation; or a still unrecognized multitude of cellular insults.

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PATHOLOGY. By Peter A. Herbut, M.D., Professor of Pathology, Jefferson Medical College, and Director of Clinical Laboratories, Jefferson Medical College Hospital, Philadelphia. Cloth. Pp. 1227, with illustrations. Price $16.00. Lea & Febiger, Washington Square, Philadelphia 6, 1955.

PRACTICAL ENDOCRINOLOGY. By Lewis M. Hurxthal, M.D., Head of Department of Internal Medicine, Lahey Clinic; in cooperation with A. Seymour Parker, M.D., Physician, Department of Internal Medicine, Lahey Clinic; and Hirsh Sulkowitch, M.D., Research Associate, Lahey Foundation. Cloth. Pp. 318, with illustrations. Price $7.00. McGraw-Hill Book Company, 330 W. 42nd St., New York 36, 1955.

THE MANAGEMENT OF OBSTETRIC DIFFICULTIES. By Paul Titus; revised by J. Robert Willson, M.D., M.S., Professor of Obstetrics and Gynecology, Temple University School of Medicine; Head of the Department of Obstetrics and Gynecology, Temple University Hospital; Consultant in Obstetrics and Gynecology and Chief of the First Service, the Philadelphia General Hospital, Northern Division. Ed. 5. Cloth. Pp. 737, with illustrations. Price $12.50. The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1955.

CLUES IN THE DIAGNOSIS AND TREATMENT OF HEART DISEASE. By Paul D. White, M.D., Consultant in Medicine, Massachusetts General Hospital; Past Clinical Professor of Medicine, Harvard University, Boston, Massachusetts. Cloth. Pp. 186, with illustrations. Price $5.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

CARDIAC EMERGENCIES AND HEART FAILURE. Prevention and Treatment. By Arthur M. Master, M.D., Cardiologist, Mount Sinai Hospital, New York, N. Y.; Associate Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University; Marvin Moser, M.D., Assistant Physician in Medicine, Montefiore Hospital, N. Y.; Adjunct Physician in Medicine (Cardiology), Grasslands Hospital, Valhalla, N. Y.; and Harry L. Jaffe, M.D., Assistant Attending Physician, Cardiology, Mount Sinai Hospital, New York, N. Y.; Lecturer in Medicine, College of Physicians and Surgeons, Columbia University. Ed. 2. Cloth. Pp. 203, with illustrations. Price $3.75. Lea & Febiger, Washington Square, Philadelphia 6, 1955.

SHEARER'S MANUAL OF HUMAN DISSECTION. Edited by Charles E. Tobin, Ph.D., Associate Professor of Anatomy, The University of Rochester School of Medicine and Dentistry. Ed. 3. Cloth. Pp. 287, with illustrations. Price $6.00. McGraw-Hill Book Company, 330 W. 42nd St., New York 36, 1955.

ANTIMICROBIAL THERAPY IN MEDICAL PRACTICE. By Harrison F. Flippin, M.D., F.A.C.P., Associate Professor of Clinical Microbiology, The Graduate School of Medicine, The University of Pennsylvania; Visiting Physician, Philadelphia General Hospital (Blockley Division); Chief, Section of Infectious Diseases, Department of Medicine, The School of Medicine, The University of Pennsylvania; and George M. Eisenberg, D.Sc., Associate in Medicine, The Graduate School of Medicine, The University of Pennsylvania; Chief, Division of Bacteriology and Immunology, Department of Laboratories, Philadelphia General Hospital (Blockley Division), Philadelphia, Pa. Cloth. Pp. 284. Price $5.00. F. A. Davis Company, 1914-16 Cherry St., Philadelphia 3, 1955.

PROLONGED AND PERPLEXING FEVERS. By Chester S. Keefer, M.D., Physician-in-Chief and Director, Evans Memorial, Massachusetts Memorial Hospitals; Wade Professor of Medicine, Boston University School of Medicine; and Samuel E. Leard, M.D., Assistant Visiting Physician and Assistant Member, Evans Memorial, Massachusetts Memorial Hospitals; Instructor in Medicine, Boston University School of Medicine. Cloth. Pp. 248. Price $5.50. Little, Brown & Company, 34 Beacon St., Boston 6, 1955.

PRACTICAL MEDICAL MYCOLOGY. By Edmund L. Keeney, A.B., M.D., Formerly: In Charge of Mycology Laboratory, Visiting Physician and Dispensary Physician (Allergy), The Johns Hopkins Hospital; Instructor in Medicine, The Johns Hopkins University School of Medicine; Fellow, American College of Physicians; Fellow, American Academy of Allergy; Member, Western Society for Clinical Research; Member, American Society for Clinical Investigation, San Diego, California. Cloth. Pp. 145, with illustrations. Price $4.50. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

SHOULD THE PATIENT KNOW THE TRUTH? A Response of Physicians, Nurses, Clergymen, and Lawyers. Edited by Samuel Standard, M.D., and Helmuth Nathan, M.D. Cloth. Pp. 160. Price $3.00 (hard cover), $2.00 (soft cover). Springer Publishing Company, 44 E. 23rd St., New York 10, 1955.

SPORTS INJURIES. Prevention and Active Treatment. By Christopher Woodard. Cloth. Pp. 128, with illustrations. Price $3.00. Max Parrish: London. Distributed by Track & Field News, P. O. Box 296, Los Altos, Calif., 1954.

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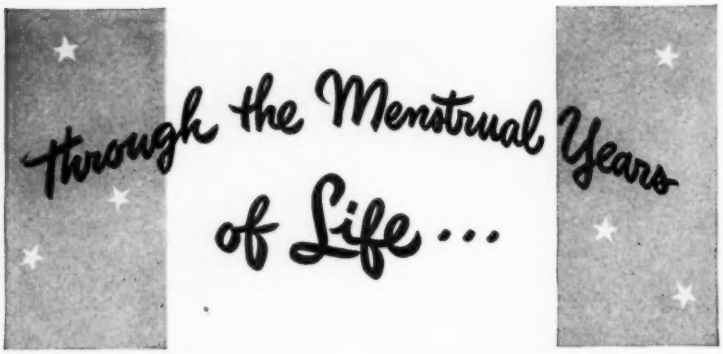

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Kareus, Lawrence Daniel
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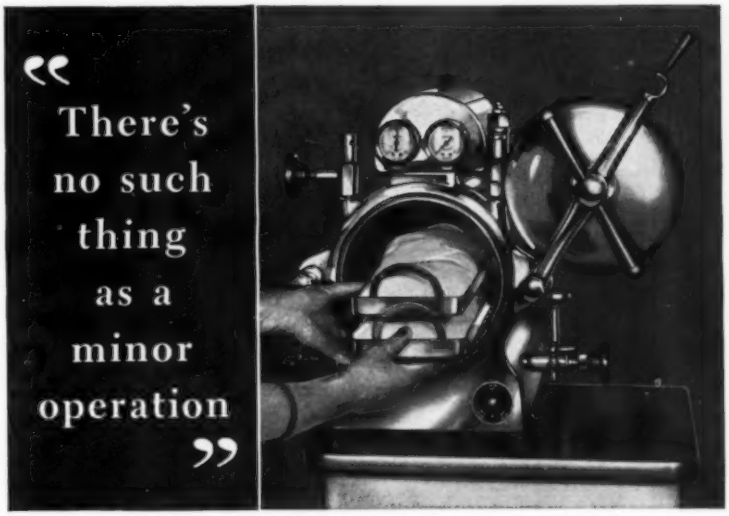

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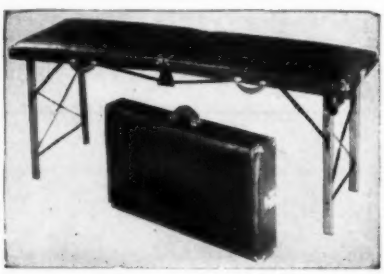

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1. Cass, L. J. and Frederik, W. S.: Malt Soup Extract as a Bowel Content Modifier in Geriatric Constipation. Journal-Lancet, 73:414 (Oct.) 1953.

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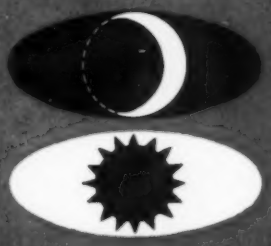

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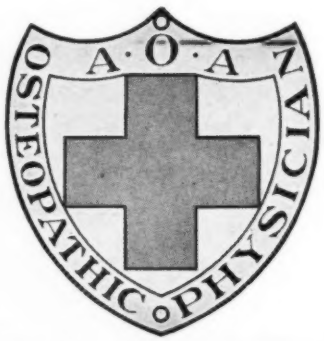

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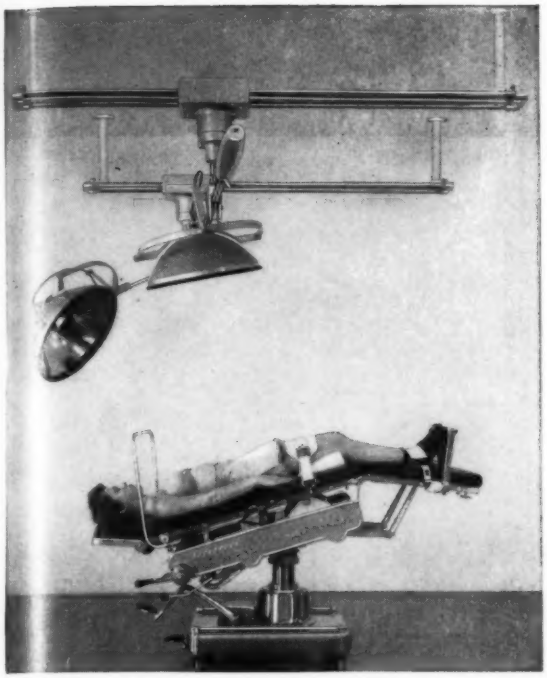

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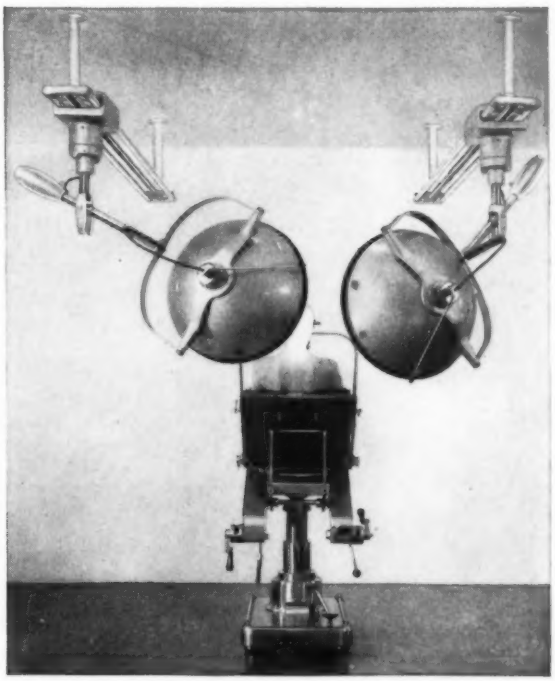

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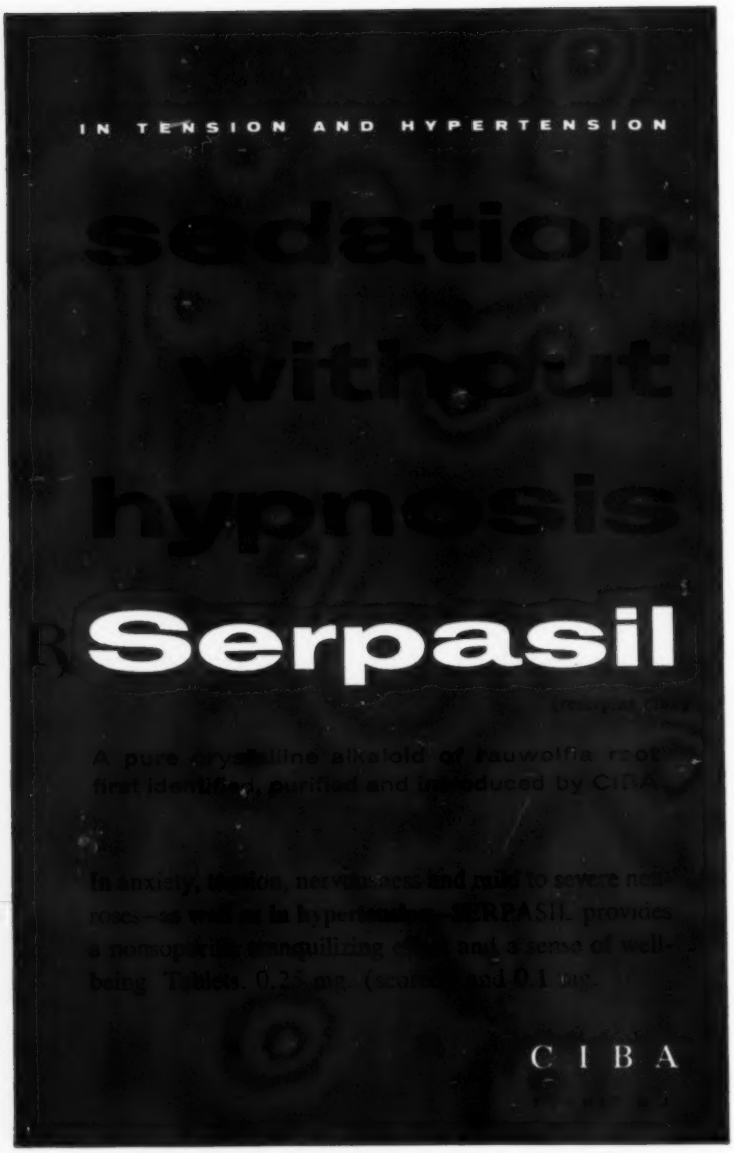

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